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EVALUATION OF REGIONAL LYMPH-NODE DISSECTION IN THE TREATMENT OF CARCINOMA*

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DISSECTION of the regional lymph nodes is carried out as a routine procedure in carcinomas of the breast, the rectum, the colon and the stomach, as part of the radical operation for dealing with the primary carcinoma. Dissections are elective procedures in the treatment of carcinomas and melanomas of the skin, and of carcinomas of the lip, mouth, external genitalia and various other organs and regions. With the object of determining the indications and value of these elective dissections, over 5000 cases of neoplastic disease have been reviewed from the recent experiences at the Collis P. Huntington Memorial Hospital, the Massachusetts General Hospital and the Pondville Hospital.

Indications for Dissection

It may be said at once that dissection is indicated when operable metastases are present. Unfortunately, this simple statement implies that the clinical recognition of the presence of metastases is accurate. However, metastases may be present before the regional nodes become obviously enlarged, and slight enlargements may have innocent causes in many cases. Enlargement of lymph nodes to a size greater than 1 cm. is strong evidence of metastasis, but there is still the possibility that the enlargement is due to inflammation rather than to metastasis. Actually, there is an error in the clinical recognition of lymph-node metastases of 15 to 30 per cent, varying with the region involved, the obesity of the patient and the care and experience of the examiner.

Because of this clinical error, it is desirable to determine — from the characteristics and the usual course of the primary carcinoma — the likelihood of lymph-node metastasis in a given case. For example, although carcinomas of the lip in general metastasized in 24 per cent of the cases, it was observed that metastases occurred in only 7 per cent when the primary carcinoma was less than 1 cm. in greatest extent, and in only 6 per cent of the cases with carcinomas of low malignancy. Similar studies of various other cancers confirmed the opinion that the tendency to form metastases is associated with long duration, large size, high grades of malignancy, an invasive as opposed to a papillary type of growth and recurrences at the site of the primary cancer. In the absence of obvious lymph-node involvement, it is only by evaluating these aspects of a cancer that one can arrive at an opinion concerning the presence of metastases.

Optimum Time of Dissection

It may be stated at this point that dissection may reasonably be deferred until metastases can be detected. Thus, fewer unnecessary dissections will be performed. When dissection is deferred until the lymph nodes are obviously involved, however, it is probable that some patients with metastases will have lost their best chance for cure as a result of the delay, and that other cases may prove to be inoperable as a result of inadequate follow-up observations. No absolute rules can be laid down regarding prophylactic, as opposed to delayed, dissection. But, when a primary carcinoma is one with little likelihood of forming metastases, in a patient who is not in sufficiently good general condition to withstand operation and who can be depended on to report faithfully for observation, it is reasonable to adopt a policy of watchful waiting. On the other hand, when there is considerable likelihood of metastasis in a patient in good general condition who may find it difficult to report regularly, prophylactic dissection is favored.

It should be unnecessary to insist that cure of the primary carcinoma is a prerequisite to any

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elective dissection. It is also a matter of experience that the mortality and complications following dissection are minimized if the dissection is postponed until after healing of the site of the primary carcinoma has taken place. Thus, the optimum time for performing a prophylactic dissection is two to four weeks after eradication of the primary carcinoma. When the lymph nodes are already involved at the time the patient is first seen, the interval may be shortened, but the dissection should still be deferred until after treatment of the primary carcinoma. When one follows a policy of watchful waiting, the patient should be examined at least at monthly intervals for a year after treatment of the primary carcinoma, and bimonthly during the second year. In these cases, dissection should be performed at once if involvement of the nodes is detected.

Extent of Dissection

The extent of a regional lymph-node dissection must be based on knowledge of the lymphatic drainage pathways from the area involved in the primary carcinoma, and the known behavior of the carcinoma concerning node metastases. For example, carcinomas of the lip and buccal mucosa rarely form metastases below the level of the omohyoid muscle, and dissections in these cases may therefore be limited to the upper neck. Carcinomas of the external genitalia may form primary metastases along the external iliac vessels as well as in the superficial nodes of the groin; it is probable that a greater number of cures would result in these cases if the radical Basset type of dissection, instead of a superficial inguinal dissection, were employed.

Bilateral dissections are frequently indicated in lesions involving the lip and the external genitalia. In lesions situated in an area of potential lymphatic drainage in more than one direction, such as the shoulder or flank, it may be necessary to carry out dissections of two areas, such as the neck and axilla, or the axilla and groin. Bilateral or multiple dissections should be performed in stages, rather than simultaneously.

Results

In the results obtained by dissection, only those cases are included in which lymph-node metastasis proved to be present. Untraced cases, and patients dying of intercurrent disease, are excluded as inconclusive. A further group is excluded in which failure resulted because of failure to control the primary carcinoma. In these cases, a dissection is obviously futile.

The results of dissection are presented in Table 1. Although these figures have no standing as an end-result report, because of the exclusions mentioned above, they probably represent what may be accomplished by regional dissections in properly selected cases when metastases are present.

TABLE 1. *Results in Cases of Cancer with Dissection of the Involved Regional Nodes.*

PRIMARY TUMOR	NO. OF CASES	CURES	
		NO. OF CASES	PERCENTAGE
Skin	36	12	33
Melanoma	38	7	18
Lip	100	58	58
Tongue	21	9	43
Mouth (other parts)	33	14	42
Vulva	14	5	35
Scrotum	10	2	20
Penis	21	11	52

These results further emphasize the fact that a large number of failures are properly attributable to local recurrence or persistence of the primary carcinoma.

It has been suggested in some clinics that the results of dissection may be improved by the employment of preoperative or postoperative radiation therapy. Although some of the patients in the present group received such treatment, there was no thorough trial of the method. In general, it has been believed that preoperative treatment postpones and complicates the dissection, and that postoperative treatment is of no benefit after a proper block dissection.

Postoperative Mortality

The figures for postoperative mortality as a result of dissection cover all cases, that is, including the figures for those in which dissection revealed no lymph-node involvement and those in which there was eventual failure through a lack of control of the primary carcinoma. When one considered the mortality incident to axillary dissection, it seemed reasonable to include patients subjected to radical mastectomy. The mortality is shown in Table 2.

Detailed analysis indicates that many of these postoperative fatalities might have been avoided.

TABLE 2. *Postoperative Mortality in Cases with Dissection.*

REGION	NO. OF CASES	DEATHS	
		NO.	PERCENTAGE
Upper neck	601	6	1.0
Neck (radical operation)	112	4	3.6
Jaw (resection)	58	5	9.0
Axilla	1224	29	2.4
Groin	260	10	3.8

One of the commonest errors consisted in operating on the primary focus of disease and the regional nodes at a single sitting. Many of the bilateral dissections were carried out in a single

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operation. It is hoped that improvements in anesthesia and operative technic, the employment of chemotherapy in cases that develop infection and the performance of multiple operations in stages rather than simultaneously will contribute to a lowered mortality.

Summary and Conclusions

A review of the results of dissection in over 5000 cases of neoplastic disease is presented.

The decision to carry out a regional lymph-node dissection in a case of cancer depends on the presence or the likelihood of development of metastases. The extent of dissection depends on the known behavior of the cancer and the anatomy of the lymphatic drainage areas. Prophylactic dissections may be performed in extensive carcinomas of high malignancy, but dissection may be deferred when there is little likelihood of metastasis, provided that the patient can be followed carefully. Control of the primary carcinoma should precede any dissection.

Dissection is capable of curing metastases in a considerable number of cases, with a postoperative mortality rate that should not be excessively high.

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DISCUSSION

Dr. Ernest M. Daland (Boston): Dr. Taylor and I are in full accord on all the facts that he has presented. I shall simply emphasize one or two points.

If one postpones a regional dissection, one should keep a very close check on the patient. We have a general rule that these patients shall report every month for the first year, every two months for the second year, every three months for the third year and so on. In private practice, one should be equally careful to see that the patients turn up and have their lymph nodes checked. For many lesions, like those on the lips, it is perfectly satisfactory to wait.

I agree that lip and neck dissections, or any other type, should be done in two stages. I have given up the idea of doing a "V" lip and a neck dissection at the same time. The majority of the lymph nodes palpated in the neck at the time that the primary lesion is seen clear up as soon as one gets rid of the primary lesion and the wound heals; in other words, they are inflammatory. If the nodes persist after a few weeks, I think that one should go ahead and do the dissection.

I do believe that a prophylactic dissection should be done in cases of malignant melanoma, when it is possible to say to which nodes the tumor would be likely to metastasize; for example, in the malignant melanomas or the extremities. I encountered a malignant melanoma in the suprasternal notch a few years ago. It was not possible to do a regional resection, since it would have meant dissection of the mediastinum and both sides of the neck, and perhaps the axilla. If one is dealing with a melanoma of the extremities, — and they are much commoner there, — one should do a radical inguinal and saphenous dissection, including the nodes along the iliac vessels; in the upper extremity, one should do a radical dissection of the axilla.

Inasmuch as Dr. Taylor's figures have proved that clinical observation is so very inadequate in revealing whether or not the lymph nodes are involved, one should not trust to palpation, but as soon as the primary lesion has been destroyed, one should go ahead with the prophylactic dissection, if necessary.

If Dr. Taylor had not explained his figures as carefully as he did, we might have been deceived by the relative values of neck dissections on lips and tongues. In the table he showed, 58 per cent of the cases with cancer of the lip and positive nodes were cured, whereas in cancer of the tongue, 43 per cent were cured. Of course, that does not represent the relative morbidity or mortality in these two types of cancer. In the former, we operate on most of the cases, unless there is very deep fixation. In the latter, we tackle really very few of them, since we must wait for the primary lesion to be destroyed. Because we fail in curing the primary lesion in many cases, and because the nodes, when we do feel them, are so far advanced, we do relatively few neck dissections. Curability of cancer of the tongue is consequently far below that of the lip.

If one does no more than cure a third to a quarter, as Dr. Taylor said in his final remarks, I believe that one will have accomplished something. If these cases were seen earlier, the number of cures for metastatic nodes would be raised considerably.

Dr. Ernest L. Hunt (Worcester, Massachusetts): At the risk of being accused of reactionism, I want to point out that my discussion deals wholly with the surgical approach and not the radiation approach.

Dr. Taylor has rendered a real service in conducting a research that turns the light of experience on the difficult problem of when and to what extent block dissections should be used to head off the extension of malignant growths, and for so concisely stating his findings and conclusions for our guidance.

In our individual experiences, I am sure that we have all made dissections and found the lymph nodes to show microscopical evidence of inflammatory process only. I am equally sure that we have deferred dissection and later had occasion to regret it.

Given a primary lesion with secondary nodes, those which seem inflammatory may harbor cancer, and those which seem cancerous may prove only inflammatory. Hence, I incline to the belief that one should consider *persistence* of more importance than *size*, and act on the principle that failure of nodes to disappear entirely within four weeks after removal of a primary focus implies malignancy. Assuming that to be so, one may well ask, Is it logical to await the resumption of activity, as manifested by increase in size of the nodes, before taking preventive action? It must also be admitted that quite sizable nodes may escape detection, even by well-trained fingers. To be sure, it is comforting to believe that secondary nodes can probably be advantageously removed even when they have attained a diameter of 1.0 cm., but is one justified in waiting for so insidious a process to attain that much momentum before acting to head it off?

Dr. Taylor does not mislead us regarding this difficulty, because he points out the 15 to 20 per cent error in recognition of lymph-node involvement. To meet that discrepancy, he directs attention to the size and grade of malignancy of the original growth. Although I am in general agreement with the precepts he defines, I wish, nevertheless, to stress the fact that a policy of watchful

waiting entails many circumstantial pitfalls that may be avoided by prophylactic dissection.

Such a pitfall is illustrated by the case of a seventy-two-year-old retired business man who regards himself as a diabetic, tests his urine frequently and is meticulous in diet. Two years ago, he left for Florida in October, conscious of a "cold sore" on his lip, about which he did not think it worth while to invite his physician's opinion, although he was careful to have his urine and blood sugar tested. He returned in May, with an ulcer on the left side of his lip and a surrounding zone of induration about 8 mm. in diameter. No lymph nodes were definitely palpable, although I thought I could feel a tiny nodule less than a rice grain in size, in the submental angle close to the jaw bone. A liberal wedge excision was done, and an excellent cosmetic result obtained. The pathological report was squamous-cell epithelioma of low malignancy. On the eve of his next departure to Florida, in October, 1940, the submental nodule could not be felt, and the lip was perfectly healed. On his return in May, a sizable node was palpable under the angle of the jaw, on the left side, and the submental nodule was definitely palpable. A suprahyoid dissection of the middle and left sides of the neck was done on May 26, 1941. The pathological report was: "Metastatic squamous-cell epithelioma of low malignancy; normal salivary gland."

Here was a very careful man, under the obsession that he was a diabetic, who, in both the primary and secondary episodes, sacrificed valuable time in relation to a lesion that threatened his life, for the sake of a sojourn in Florida to benefit his general health and diabetic tendency. Certainly, one may assume that a prophylactic dissection after the first operation would have been of greater value to him than one done a year later after the growth had become well implanted and actively growing in a node far around under the angle of the jaw.

If removal of regional nodes is accepted as obligatory in cancer of the breast, does not the same principle apply to the lip or vulva, allowing of course for the infection inherent in the situation of the orificial growths?

Considering the nature of cancer, I wonder if we do not rely too naïvely on this matter of grading. I have been asking myself the questions: Does the degree of malignancy depend entirely on the histologic structure of the growth? May not a growth acquire a higher degree of malignancy according to environmental factors, such as persistent irritation, infection, diet and perhaps hereditary factors of susceptibility and immunity? In the case cited above, a growth of low malignancy histologically had a lively invasive quality.

I conclude by voicing the following convictions: When in doubt whether a prophylactic dissection is indicated, and in the absence of serious contraindications, the correct principle is to err on the side of thoroughness and do the dissection as soon as it may be done safely, rather than to be driven to it at some future and less favorable moment.

Too great reliance should not be placed on grading in justification of a negative attitude toward preventive surgical treatment.

Dr. David W. Parker (Manchester, New Hampshire): I agree entirely with Dr. Taylor's conclusions. I therefore do not wish to take up time with discussion, but should like to report briefly a case of squamous-cell carcinoma in a thirteen-year-old boy, which, I think, is worthy of record.

I saw this child for the first time a few days prior to July 15, 1941. A small nodule or papule had appeared on the inner surface of the upper third of the right arm three or four months previously. He thought that it was probably an infection, and squeezed it. It did not disappear, however, but grew progressively larger. In the middle of June, he consulted a doctor, who thought it was a cyst and opened it. He expressed some bloody fluid, but a sinus persisted and did not heal. There was no pain, but there was localized redness and thickening. On July 15, I excised the nodule and sent it to Dr. Ralph Miller, in Hanover, for examination. The pathological report was as follows:

> The specimen consists of a partially formalin-fixed gray-white red-brown, roughened piece of skin, 1.0 by 0.8 cm., covering a flattened gray-brown and red-brown piece of tissue, 0.8 by 0.7 by 0.3 cm., which, on section, reveals a cavity 0.5 by 0.8 by 0.2 cm., filled with soft, dark, brown-red material. Microscopical examination reveals anaplastic squamous epithelial cells lying beneath normal stratified squamous epithelium, with clear cytoplasm, in which are many areas of hemorrhage. Pathological diagnosis: squamous-cell carcinoma (Grade II).

I was unable to find any palpable nodes in the axilla. However, because a definite percentage of involved axillary nodes are not palpable, — a point that has been brought out so effectively in Dr. Taylor's paper, — excision of the axillary contents seemed advisable. This was done on July 24. The pathological report was as follows:

> Examination reveals normal lymph nodes. There is no evidence of malignancy. In view of the low degree of malignancy of the original tumor, and the absence of any evidence of it in the surrounding tissue, the prognosis is excellent.

In this case, there was no involvement of the axillary contents, yet I am firmly convinced that the procedure was definitely justified, and should be carried out as a matter of routine in lesions of this type.

Dr. Taylor (closing): I was afraid that I should upset certain of the orthodox brethren by not coming out more strongly for prophylactic dissection, and I am very grateful to Dr. Hunt for making the point that, if one is in doubt, dissection should be performed. I did not emphasize enough, perhaps, the absolute necessity for control of the patient, if any deferred program is contemplated and dissection is not offered as part of the initial treatment. The difficulty with Dr. Hunt's patient who went off to Florida is the same thing in a higher economic bracket that we see in the clinic when a patient comes to Boston from the outposts of civilization; once he gets back there after the carcinoma is treated, one knows perfectly well that he will never come out of the woods again until he has a massive recurrence. Dr. Hunt's patient would not come off the beach until he had a massive recurrence.

The patient who cannot be controlled and seen at frequent intervals should have the benefit of a prompt regional dissection. How much damage is done by deferring the dissection and waiting until the nodes appear, I cannot say. I tried to determine that in relation to cancers of the lip. Only 33 per cent of the patients in whom the dissection was deferred for longer than six months from the time the lip was operated on were cured, in contrast to those in whom the dissection was done at once, in whom, even in the presence of positive nodes, the cure rate was about 60 per cent.

I think that the group deferred for more than six months contained a good many neglected cases or cases in which the best program was not followed. There may be an optimum time when, even with deferred dissection,

In 14 cases of acute peripheral neuropathy, pyruvic acid curves following glucose ingestion were abnormally elevated and prolonged. The maximum rise in blood pyruvate was not only greater than that seen in normal subjects, but the elevation above the fasting level was maintained for at least four hours. Furthermore, the maximum rise did not occur at the end of the first hour, as in normal persons, but the pyruvate continued to rise in every case, and maximal figures occurred at the end of the second, third or fourth hour. Particular mention must be made of one case of acute peripheral neuropathy with a normal fasting blood pyruvate, and a markedly abnormal pyruvate curve following glucose ingestion. These curves have recently been published elsewhere.[18]

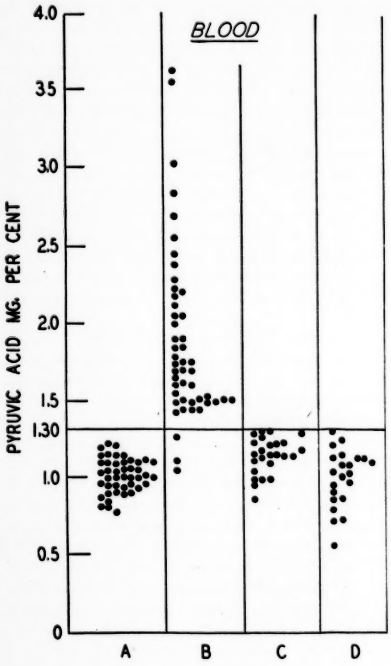

FIGURE 1. *Determinations of Blood Pyruvic Acid.*

A — *Normal subjects;* B — *acute peripheral neuropathy;* C — *chronic peripheral neuropathy;* D — *chronic alcoholics without any objective evidence of nutritional deficiency.*

DISCUSSION

It will be noted that in 45 of 48 cases of acute peripheral neuropathy, the blood pyruvate was elevated. Two of the three subjects with normal fasting blood pyruvates showed no response to thiamin therapy; as a matter of fact, they grew progressively worse, despite very adequate nutritional therapy. These were the only cases in the entire group that showed no response to vitamin therapy. In the third case, the normal fasting blood pyruvate was associated with a marked abnormality in the pyruvic acid curve following glucose ingestion; the blood pyruvate was of the type that we have seen in 14 cases of acute peripheral neuropathy thus far studied. This may indicate that the pyruvic acid curve following glucose ingestion is a more sensitive index of thiamin deficiency than the fasting blood pyruvate is.

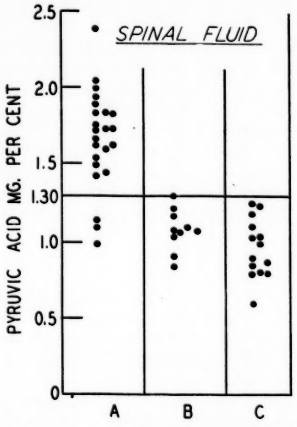

FIGURE 2. *Determinations of Cerebrospinal-Fluid Pyruvic Acid.*

A — *Acute peripheral neuropathy;* B — *chronic peripheral neuropathy;* C — *chronic alcoholics without any objective evidence of nutritional deficiency.*

In 3 of these cases, the urinary keto acid excretion after the ingestion of glucose was studied and found to be within normal limits. During this period, the pyruvic acid curve was abnormally elevated and prolonged. This confirms Waelsch's[19] evidence that the origin of urinary keto acid is not related to the carbohydrate intake. It also indicates that urinary pyruvate levels cannot be used as evidence of deficient pyruvic acid metabolism in man.

We wish furthermore to call attention to the fact that these cases of acute peripheral neuropathy are invariably associated with some type of disturbance in cortical functioning.[20] This varies from simple disorientation with memory gaps, through delirium tremens, the Korsakoff syndrome and the Wernicke syndrome.

The Wernicke syndrome is invariably associated with a thiamin deficiency,[21, 22] but evidence has been presented that it is usually the result of a multiple nutritional deficiency.[22] The other encephalopathic syndromes may also be related to specific nutritional deficiencies, but the evidence presented to date is not conclusive.[23]

It is also essential to emphasize the fact that the modalities most seriously involved (touch, position and vibration) in the peripheral neuropathy of alcohol addicts are those which are eventually to be carried in the dorsal columns of the spinal cord. Alcoholic patients frequently show dorsal sclerosis at post-mortem examination. Clinically, the loss of position sense, vibration sense, light touch and plantar dysesthesia may all be duplicated by lesions in the dorsal columns of the spinal cord.[24, 25] It is therefore virtually impossible to tell whether or not any case of peripheral neuropathy in the alcohol addict is complicated by dorsal sclerosis.

These cases not only show involvement of the peripheral nerves, but also are invariably complicated by disorders in cerebral functioning and perhaps also in the functioning of the dorsal columns of the spinal cord. This becomes readily explicable if one realizes that the brain[26] and spinal cord[27] utilize carbohydrate as their preferential foodstuff, and that this utilization is necessarily interfered with if certain enzyme and coenzyme systems necessary for the proper breakdown of carbohydrate are lacking.

The patients with chronic or residual peripheral neuropathy showed normal figures. These cases include those who had received adequate vitamin therapy without a complete return to normal functioning of the peripheral nerves and persons who had been on hospital diets for some time. It is possible, however, that many of the latter still show an inability to catabolize pyruvic acid properly, under the additional stress of metabolizing ingested glucose. Such studies are now under way. It has been previously suggested that in cases in which thiamin therapy had not been completely successful, irreversible pathologic changes had occurred.[28] These cases in which the metabolic upset had been corrected but nerve involvement persisted seem to confirm this suggestion. On the other hand, such cases may actually have a certain amount of dorsal sclerosis, and it is quite possible that thiamin therapy is of no help. Finally, many of these cases of so-called "irreversible" peripheral neuropathy frequently show marked improvement if a diet rich in other nutritive essentials is added to the thiamin therapy. Knowledge of normal nerve metabolism is by no means complete, and as additional light is shed on these other factors, it may be that certain cases of irreversible peripheral neuropathy will prove to be completely reversible.[29]

In the 22 cases of alcoholism without involvement of the central or peripheral nervous system, — and without any other evidence of nutritional disturbance, — the pyruvic acid levels in the blood and spinal fluid were normal. These results indicate that elevations in blood pyruvate are related to the nutritional disturbances that complicate chronic alcoholism and not to chronic alcoholism as such.

In addition, we[30] have studied 280 cases of various neuropsychiatric and medical disorders unassociated with any clinical evidence of thiamin deficiency, and all showed normal levels of pyruvic acid in the blood. On the other hand, we have noted hyperpyruvemia in 22 of 35 cases complicated by marked elevations in temperature and unassociated with any clinical evidence of thiamin deficiency. In these, the elevation in total metabolism may be a result of prolonged fever, which so increases the thiamin requirements of the body that a deficiency occurs, and hyperpyruvemia results. On the other hand, it must be noted that none of these cases had peripheral neuropathy. This suggests either that the metabolic disturbance must exist for some time before peripheral neuropathy occurs or that hyperpyruvemia may be related to other metabolic disturbances in addition to thiamin deficiency. We also wish to emphasize the fact that we do not believe that a normal fasting pyruvate level invariably constitutes evidence of thiamin adequacy, and we recommend that when doubt exists, the patient be subjected to the additional stress of metabolizing ingested glucose. An inability to metabolize this substance properly is further evidence of a deficiency in thiamin.

Our results suggest that acute peripheral neuropathy in the alcohol addict is invariably associated with a deficiency in thiamin. It must be understood, however, that this does not necessarily mean that a deficiency of thiamin is the only cause of peripheral neuropathy in the alcohol addict, and we are quite certain that, in some cases, recovery is markedly expedited if other factors of the vitamin B complex are given in addition to thiamin.

As a matter of fact, Wortis, Wortis and Marsh[31] noted that 18 such patients showed a marked diminution in the vitamin C levels of the blood and spinal fluid. The authors did not believe that vitamin C deficiency was causally related to the peripheral neuropathy, but stated that these findings indicated the importance of nutritional factors in the nervous and mental disorders that complicate chronic alcoholism.[32]

Our results, however, seem to indicate very clearly that acute peripheral neuropathy in the alcohol addict is associated with hyperpyruvemia. One of the recognized causes of hyperpyruvemia is thiamin deficiency. These cases with hyperpyruvemia invariably respond to thiamin therapy with a reduction of pyruvic acid levels to normal. This correction of the metabolic defect is usually associated with clinical improvement, but rarely with clinical cure.

Summary and Conclusions

Patients with "acute" peripheral neuropathy almost invariably show hyperpyruvemia. This is not true of patients with "chronic" peripheral neuropathy, or of patients with chronic alcoholism unassociated with nutritional disturbances.

In 14 cases of acute peripheral neuropathy in the alcohol addict, the pyruvic acid curve following glucose ingestion was abnormally elevated and prolonged.

Disturbances in cerebral functioning, and possibly in the functioning of the spinal cord, are invariably associated with acute peripheral neuropathy.

The relation of hyperpyruvemia to cases of acute peripheral neuropathy suggests that this disease is associated with a deficiency of thiamin. On the other hand, data are presented to indicate that other nutritive essentials may well play a role in the picture as it is usually seen.

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REGIONAL ANESTHESIA: ITS USE IN OBSTETRICS AND GYNECOLOGY*

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A SERIOUS and circumspect view of the physiologic considerations that surround the production of anesthesia and the anesthetic needs of a patient inevitably impresses one with the indispensability of regional anesthesia.

Before the methods to be used are considered, it may not be amiss to recall that they all require definite anatomic knowledge, and that generally osteologic prominences serve to localize the approaches to the nerves and help visualize the paths they traverse. A knowledge of the peripheral distribution of the nerves supplying the operative field and the involved structures is equally essential in deciding the optimum injection points. For, obviously, the right nerves must be reached in the best location, with an adequate amount of anesthetic solution, to produce a satisfactory type of anesthesia.

SENSORY NERVE SUPPLY

The female external genitalia receive their sensory nerve supply from the pudendal plexus, through the pudic nerves and the pudendal branches of the small sciatic nerves, which supply the larger part of the vagina, the labia minora and the posterior half of the labia majora. The anterior portions of the labia majora are innervated by the terminations of the ilioinguinal and genitocrural nerves, and infrequently by the iliohypogastric, which originate in the first two lumbar nerves. The perianal tissues and skin are also served by the coccygeal plexus.

The pudendal plexus is formed by the anterior collateral branches of sacral plexus, the fourth and fifth sacral nerves and the coccygeal nerves, in addition to sympathetic-system filaments. The pudendal plexus is really an inferior continuation of the sacral plexus. It divides into visceral and muscular branches, and in it originate the small sciatic and pudic nerves and sacrococcygeal plexus. The visceral branches unite with the hypogastric plexus and sympathetic filaments to form the pelvic plexuses supplying the bladder, the rectum, and the cavernous and uterovaginal plexuses.

The muscular branches, which supply the levator ani, coccygeus and external sphincter ani, afford sensory fibers to the skin over the tip of the coccyx and base of the ischiorectal fossa.

The small sciatic nerve is entirely sensory, arising from the first, second and third sacral nerves, and leaving the pelvis through the great sacrosciatic foramen, runs to the midthigh, posterior to the great sciatic nerve.

The inferior pudendal nerve leaves the small sciatic nerve at the lower margin of the gluteus maximus muscle, curves below the ischial tuberosity, and passing into the fold between thigh and perineum, enters the deep fascia lateral to the ischiopubic ramus. From there, its terminal branches go to the labia majora and clitoris.

The pudic nerve originates from the second, third and fourth sacral nerves, emerges from the pelvis through the greater sacrosciatic notch, rounds the ischial spine, and re-enters the pelvis through the small sacrosciatic notch. It immediately gives off the inferior hemorrhoidal nerve, and enters Alcock's canal, a fascial compartment on the medial aspect of the obturator internus muscle. It emerges at the inner and forward aspect of the ischial tuberosity and, at the base of the triangular ligament, divides into its terminal branches, the internal and external perineal nerves and the dorsal nerve of the clitoris. The pudic nerve, therefore, supplies the perianal skin and sphincter, the perineum and part of the labia majora, the perineal and external urethral muscles and the clitoris.

The sacrococcygeal plexus, formed by the fourth and fifth sacral and coccygeal nerves, supplies the pericoccygeal skin and tissues forward to the anus.

Thus, it is seen that the nerve supply of the external genitals is derived mostly from the anterior collateral branches of the sacral plexus and the pudendal plexus (lower sacrals) and the coccygeal nerves, with sympathetic-nerve contributions. The lumbar plexus contributes through the ilioinguinal, genitocrural and occasionally the iliohypogastric nerves. To effect anesthesia, one must block all these nerves.

METHODS OF REGIONAL ANESTHESIA

The region may be anesthetized in several ways: by intraspinal block, the procedure of choice in all fat and difficult women; by sacral block; and by perineal field block.

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Intraspinal Block

It is not my purpose exhaustively to discuss spinal anesthesia, but a few simple facts concerning it may be worth recalling. It differs from other types of central nerve block in that it is subdural rather than extradural. Induction requires puncture of the dura mater and subarachnoid placement of the anesthetic solution. There must be a minimum of local trauma in introducing the anesthetic. This means proper position of the patient, the use of a good flexible needle of small gauge, and avoidance of contact with the periosteum and, especially, with the intervertebral disk and nerve radicles. Adherence to these rules resolves itself into experience and the knowledge of anatomy. Bad results are generally due to lack of fundamental knowledge and inept technic.

Once placed, the anesthetic and its effects are largely beyond control. Hence, the amount and placement level must be conditioned by the patient and the operation to be done. For abdominal, gynecologic and obstetric operations, the level is the third space, with 100 to 120 mg. novocain diluted in spinal fluid to a volume of 3 or 4 cc. In a prolonged case, 8 to 10 mg. of Pontocaine is added. For vaginal work and deliveries, 50 mg. of novocain is similarly diluted and placed intrathecally in the fourth space. The patient is kept flat on the table, or a moderate Trendelenburg position may be used if the patient is not pregnant or if there is no large abdominal tumor, intraperitoneal fluid or marked distention. Ephedrine sulfate, ¾ gr. hypodermically before induction of anesthesia, lessens blood-pressure fall, although this in itself is unimportant provided the pulse remains good and the respirations are not embarrassed. If respiratory embarrassment occurs, immediate inhalation use of oxygen under pressure and intravenous fluids are invariably effective. I have given over three thousand spinal anesthesias, with but one death that could be ascribed to the anesthetic; I admit, however, a few close calls. In the Margaret Hague Maternity Hospital, we have administered over five thousand spinal anesthetics in the past eight years, with three deaths that could be ascribed, with any reason, to the type of anesthetic given. It scarcely needs saying that we should have long since given it up had we found the dangers even remotely as present as its antagonists contend. The vast majority of the detractors of the procedure have had little or no experience with it and possess an inferior technic. We do not claim it to be the only anesthetic to use or the best in every case, and it has definite contraindications. But it is a very good and very safe anesthetic when properly employed, and its use is not infrequently the major factor in a patient's recovery from a difficult plight.

Sacral Block

Unfortunately, the facility of administering a spinal anesthetic has obscured the value of another form of nerve block — trans-sacral, with caudal, block. This is a perfectly safe type of anesthesia that is only moderately difficult to employ. The combination has the virtues of subdural block, with none of the hazards. The objections are the time it takes, frustrating fat and, in pregnancy, inability to obtain the optimum induction position.

Although I admit that sacral block has taken a poor third place to spinal anesthesia and local infiltration in pelvic, perineal and rectal work in my own hands, it is a good type to have in reserve for special conditions. For cases of hypertensive cardiac disease or pre-eclamptic toxemia with respiratory infection, or in any other case in which a spinal anesthesia might be somewhat hazardous or the patient is suffering with an upper respiratory infection of any degree, a sacral block is ideal.

The technic may be rather simply and quickly set down, thanks to the pioneering work of Labat[1, 2] and others,[3-7] who have so well standardized its application. If the prone position, with a cushion under the patient's hips to raise the sacrum, is not possible, the next best one is the extreme Sims. Caudal block is first induced, since it takes about twenty minutes for it to be effectively obtained and because it fails in 10 to 20 per cent of cases. For caudal block, the puncture is made in the sacral hiatus in the middle of a triangle formed by the sacral cornua and the fourth sacral spine. The sacral hiatus is palpated with the finger, after which the needle is introduced with the bevel upward at an angle of 20° with the skin. The needle passes through the ligament and forward to the anterior wall of the canal; it is then retracted and depressed 20° farther. It is then introduced as far forward as a line joining the posterosuperior iliac spines. This marks the average lower depth of the dural-sac level. It is essential not to carry the puncture farther, and not to introduce the solution if spinal fluid is then obtained. Thirty cubic centimeters of a 2 per cent novocain solution is injected or if parasacral block is also employed, it is better to use 30 cc. of a 1 per cent novocain solution. I generally add about 5 minims of epinephrine solution, about 0.5 mg. to each 100 cc. of anesthetic employed.

Paravertebral sacral block may then be proceeded with. The landmarks here are the sacral cornua and the posterosuperior iliac spine. As a matter of fact, the point most commonly chosen for the

latter is the regional posterior spine, which is the last prominence at the posterior extremity of the iliac crest, usually about 1 cm. above the anatomic spine. The sites of injection are chosen as follows: wheals are raised 1 cm. medial to and below the regional posterior spine, with another wheal lateral to and just above the sacral cornua; a line, which is drawn between these two points and beyond the posterosuperior spine, is divided between the two chosen points into three equal parts, with two wheals of novocain. The ilial or uppermost wheal marks the aperture of the second sacral foramen. The cornual wheal marks the site of the fifth sacral foramen. The other two wheals, marked out between these, locate the third and fourth sacral foramens. The first sacral foramen is punctured through a wheal raised 3 cm. on the line above the ilial or first wheal. The manner of injection is as follows: The bone is met on the dorsum of the sacrum before the foramen is perforated, and the needle is then passed in for varying distances. It is probably easiest to remember that from the first to the fifth foramens, respectively, the distances that the needle enters after touching the bone diminishes by 0.5 cm. — from 2.5 to 0.5 cm. — for each foramen, and the amount of novocain, a 1 per cent solution being used, diminishes from 6 to 2 cc., being reduced 1 cc. for each foramen. This, then, permits the introduction of 20 cc. of novocain solution on each side of the sacrum in contact with the sacral nerves as they emerge from the anterior surface of the sacrum; the radicles of the sacral and perineal plexuses are thus blocked. Anesthesia here follows very promptly, and by the time of its completion, caudal anesthesia is generally effective if it is to become so.

Perineal Field Block

There are several effective ways of obtaining a satisfactory perineal field block; the one that I routinely employ, which has proved satisfactory, is not claimed to be better than any one of the other methods used, but it has repeatedly given and continues to give satisfactory results.

The principle employed is the blocking of nerves supplying the vulval and perineal region as they approach that area. The main injection involves blocking the pudic nerve as it emerges from Alcock's canal near the ischial tuberosity. The other nerves are blocked as their terminations approach the field. The first injection is made through a wheal raised over the ischial tuberosity. A 10-cm. needle is introduced, and bony contact is first made with the ischial tuberosity; it is then passed 2 cm. medial to and 2 cm. deeper than the tuberosity. This is approximately the point at which the pudic nerve emerges from Alcock's canal. Fifteen cubic centimeters of a 1 per cent solution of novocain, with ephedrine, is injected at this site on both sides. The needle is then withdrawn, reintroduced and conducted subcutaneously about 0.5 to 1 cm. deep across the perineum to the ischial wheal on the opposite side. It is then withdrawn and directed from both wheals backward toward the anus until injections are made into the sphincter muscle itself. Reintroduced through the ischial wheals, the needle is directed obliquely inward toward the introitus, then deflected sharply upward along the inner aspect of the vestibular bulb and under the labia minora, distending the structures with novocain as far forward as the clitoris. Thus, a diamond-shaped block of the introitus is evolved. The transverse bi-ischial infiltration with the anterior limbs blocks the ilioinguinal, iliohypogastric and genitocrural nerves, the pudendal branch of the small sciatic nerve and, partly, the internal and external branches of the pudic. The posterior arms block the hemorrhoidal branches of the pudic nerve and the terminations of the anococcygeal plexus, and paralyze the anal sphincter. Direct infiltration of the pyramidal perineal body permits immediate, deep perineotomy.

When the innervation of the external genitals is recalled, it is apparent that not only the sensory but also the muscular nerves of these structures are effectively blocked, and anesthesia is produced for a third or half of the way up the vagina. One must also remember that, even with an outlet-forceps application, the blade tips are far in the vagina, where there is less sensation but also no anesthesia, and anesthesia should therefore be supplemented with deliberate and gentle movements. For deep vaginal work in gynecology, submucosal local infiltration is added, as well as a broad ligament block for cervical procedures, to anesthetize the uterine and vaginal sympathetic plexuses. It is recommended that adrenalin always be added to the anesthetic solution. In obstetrics, the extreme vascularity of the parts causes a rapid washing out of novocain, with anesthetic failure, unless this precaution is taken.

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MEDICAL PROGRESS

HEMATOLOGY: DISEASES OTHER THAN ANEMIA

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BONE MARROW

THE sternal bone-marrow biopsy, performed chiefly by means of the simple puncture technic, has become almost a routine hematologic procedure. Not only is it of value in the differential diagnosis of so-called "refractory anemia," "pancytopenia"and so forth, but it is distinctly a necessary maneuver in ruling out leukemia in cases of both thrombopenic purpura and hemolytic anemia, especially when splenectomy is being seriously considered. The physician who familiarizes himself with the technic will gradually develop a better understanding of the physiologic pathology underlying the various blood pictures. Since the latter may give only an imperfect reflection of what goes on in the blood-forming tissues, careful study of the marrow should be of real help in developing a fundamental knowledge of the blood conditions. To learn "to walk about" in the marrow requires only a little instruction but a good deal of practice and patience. In any event, interpretation of bone-marrow pictures is not nearly so difficult as it seems at first glance.

Probably the best monograph thus far on the bone marrow (in German) is that by Rohr,[1] of Zurich, Switzerland.

Recently, the sternal bone marrow has been utilized for therapeutic purposes. Thus, Morrison and Samwick[2] cite the case of a patient with aplastic anemia who was given several injections of normal sternal marrow, totaling 13 cc., obtained from compatible donors. The patient recovered. It is to be noted that just prior to the bone-marrow "transfusion," the patient's blood showed well-defined evidences of increased regenerative activity on the part of the marrow, that is, marked polychromatophilia and the presence of nucleated red cells. Frequent reticulocyte and platelet counts were not performed. Various other forms of therapy, such as liver extract and iron, were also used. It is highly doubtful whether the few cubic centimeters of actual normal bone-marrow tissue injected into the sternum had any effect on an organ that approximates about 1400 cc. in volume.

Fluids and blood injected into the sternal marrow cavity quickly reach the venous circulation, as demonstrated by Tocantins and O'Neill.[3] These investigators, after a thorough study of the sternal marrow in various age groups, devised a special needle and adapter by which, in an emergency, it is possible to transfuse into the sternal cavity large quantities of blood or saline solution. This might conceivably be important in a case with extensive burns or when the veins are collapsed or inaccessible.

FRAGILITY TESTS

The methods in general use for determining hypotonic fragility are quite inaccurate. The method most commonly employed, that of Giffin and Sanford, utilizes unmeasured drops of blood, which are placed in tubes containing only roughly estimated amounts of hypotonic saline solution. In the procedure of Daland and Worthley,[4] the solutions and the amount of blood are accurately measured. Furthermore, the blood is adjusted to constant volume so that even in the presence of severe anemia, approximately the same number of red cells are added to similar volumes of saline solution. Even this method, however, leaves room for improvement, since the tube in which beginning hemolysis is present is often difficult to define and the other readings are also more or less subjective. To avoid these disadvantages, Waugh and Asherman[5] used the Evelyn photoelectric colorimeter and measured the degree of hemolysis directly. By this method, curves of erythrocyte fragility measuring the percentage of hemolysis with the various solutions of hypotonic saline could be drawn. In a later paper, Waugh and Lamontagne[6] illustrated an application of this method in the study of the changes in fragility following splenectomy in congenital hemolytic jaundice. Hunter[7] has recently described a similar method, and Cruz and his co-workers[8] also utilized this method in a unique study with radioactive iron of the effect of age on the susceptibility of the erythrocyte to hypotonic saline solutions. The latter authors injected radioactive iron into dogs rendered anemic by

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bleeding. This "tagged" iron is present only in the young red cells, — reticulocytes, — and it was thus thought that an accurate determination of the relative fragility of older as against younger red cells could be made. The surprising result was obtained that the very new red cells in the circulation were "markedly less resistant" to hypotonic saline solutions than the older cells. Unfortunately, this type of experiment probably does not yet completely solve the vexing question whether younger or older red cells are more fragile.

Blood Volume

Gibson has been in great part responsible for perfection of the blood-volume test that utilizes the Evelyn photoelectric colorimeter and the Evans blue dye. In a recent article,[9] a general review of his findings is presented. In the diagnosis and follow-up of a case of polycythemia, determination of the blood volume is often of great value. In studies of hemolytic anemia, the relation of the total mass of hemoglobin to the urobilinogen content of the feces gives an index of blood destruction that is at times of diagnostic value.[10] Although the Gibson–Evelyn method for determining the blood volume is universally conceded to be the most accurate, Ebert and Stead[11] have recently criticized it because the cell-volume reading depends on the hematocrit and this factor is difficult to determine accurately. The latter investigators showed that the relative proportion of red cells varied considerably in different blood vessels of the body, and that the basing of the hematocrit on determinations made from basilic veins might lead to error. This has, of course, been well known, but is difficult to get around, unless some such method as that of Barcroft (utilizing carbon monoxide) is used.

Hahn, Ross and their co-workers[12] have devised another interesting method, which has not yet been applied to human beings.* From a dog injected with radioactive (tagged) iron, a known amount of blood is injected in other dogs, and the blood volume calculated from the relative amount of radioactivity present in the injected dog's blood.

Tests of Bleeding Time

The test in use for many years is that of Duke, in which a puncture of the ear is made, the resulting wound being blotted every thirty seconds. Ivy, Shapiro and Melnick[13] attempted to make the test more accurate by pricking the skin of the forearm under conditions of venostasis — a blood-pressure cuff being inflated over the arm to a pressure equivalent to 40 mm. of hemoglobin. In a more recent article, Ivy, Nelson and Bucher[14] report on the standardization of the various factors of pressure, type of puncture, type of blade used and so forth. By this method, the bleeding time varies between sixty-one and two hundred and forty seconds.

*Dr. J. F. Ross of the Evans Memorial, Massachusetts Memorial Hospitals, has recently applied this method to human blood-volume determinations by injecting tagged red cells from previously injected polycythemic patients into other persons.

Blood Groups

Rh Factor

One of the outstanding examples of progress in hematology during 1941 has been the development of knowledge concerning the Rh factor. Landsteiner and Wiener[15] showed in 1940 that by the injection of blood cells of the monkey — *Macaca rhesus* — into rabbits, an antirhesus red-cell agglutinin was built up in the rabbit serum. Curiously enough, it was found that this agglutinin (called "anti-Rh") also agglutinated the red cells of most human subjects, without reference to the regular human blood groups (O, A, B or AB). Further studies by Wiener and Peters[16] demonstrated that the cause of certain severe blood-transfusion accidents, involving donors and recipients of the same blood group, lay in the presence of this anti-Rh agglutinin in the recipient, reacting against the donor's susceptible red cells, which contained an Rh binding substance or agglutinogen; such persons are said to be "Rh+." Levine and his co-workers[17] demonstrated that approximately 85 per cent of 1035 persons were Rh+, and 15 per cent Rh–. Thus, if an Rh– subject (that is, one having no Rh agglutinogen in his red cells) is given blood from a donor with Rh+ cells, a reaction will occur if the recipient's serum contains the anti-Rh agglutinin. Only an Rh– person can possess anti-Rh agglutinin in the serum, since the two substances are mutually incompatible. It was further found that most of the transfusion reactions involving the Rh factor occurred in women, either pregnant or in the puerperium, who had been given blood from their husbands. Analysis of several of these cases by Wiener and Peters[16] and Levine and his co-workers[17] demonstrated that the recipient was Rh– but had an anti-Rh factor in her serum, and that the husband donor was an Rh+. The possibility was suggested that the mother might have become immunized against her husband's red cells by way of the fetus (which usually was Rh+). The concept of isoimmunization in the human being by the Rh factor was thus developed.

Shortly thereafter, Levine, Katzin and Burnham[18] pointed out that in cases of erythroblastosis foetalis, the following combination of facts was present: the mother was Rh– and often showed

anti-Rh agglutinin in her serum, the father was Rh+, and the infant was Rh+. It was suggested by these workers, and borne out by analysis of repeated cases, that some Rh– mothers might become immunized to the Rh factor if the baby's red blood cells passed into the general circulation via the placenta. When this had taken place, an agglutination reaction might occur within the placental tissues, damaging the red cells and producing the clinical picture of erythroblastosis foetalis in the newborn infant. This rather startling although attractive theory of isoimmunization as an explanation of the pathogenesis of the disorder has already aroused a great deal of interest and may be of considerable practical significance.

To test a subject for the presence or absence of Rh factor in the red cells, it is necessary to have anti-Rh testing serums. These are not now readily prepared, and reliance must be placed on human subjects who have developed an anti-Rh substance in the serum. Since these are relatively scarce and since, furthermore, the actual testing is somewhat more delicate and "tricky" than ordinary blood typing, it becomes essential for the practicing physician to observe the following rules regarding transfusions of blood. In the first place, compatibility tests, particularly with patients who are pregnant or in the puerperium, or who have had previous transfusions, should be performed by the Landsteiner test-tube technic. One drop of the serum to be tested, one drop of a 2 per cent red-cell suspension and one drop of physiologic saline solution are placed in small test tubes, incubated at 37°C. for thirty to sixty minutes, and centrifuged for one minute at slow (500 RPM) speed, and the sediment of the tube is inspected[19] and the tube observed for clumps — a definitely positive reaction is readily observed with the naked eye, and a negative reaction should be checked by microscopic examination of the sediment. Secondly, if a transfusion is required in pregnancy or the puerperium, it is best not to use the husband as a donor. And, finally, in erythroblastosis foetalis, one should go to great lengths in obtaining an Rh– donor for the infant, and in performing exceedingly careful cross-matching tests preliminary to transfusions; the infant's mother should not be used as a donor. The further general suggestions might be made: before a transfusion is given, the question whether it is really necessary should be asked, and typing and cross-matching should not be performed by the most inexperienced junior on the service. Correct typing and cross-matching before a transfusion are important tests and deserve the utmost care; they are best done by competent, unhurried workers.

Miscellaneous Data

Group A (Type II, [illegible]) is known to be made up of Subgroups A_1 and A_2. With the use of high-potency testing serums cells of Group A or Group AB can almost always be distinguished. However, occasional persons of Group A or AB react feebly even to the most potent testing serums and may therefore be miscalled Group O or B. Recently, Wiener and Silverman[20] demonstrated that a third A agglutinogen (A_3) was occasionally present, and might be confusing in determining the blood group. This brings up another possible cause for transfusion reactions, which can be avoided only by very careful compatibility tests.

The preparation of anti-M, anti-N, anti-A and anti-B testing serums is discussed by Silverman.[21] The difficulties involved in preparing a potent anti-N serum are pointed out. The use of M and N testing serums in the exclusion of paternity is increasing as knowledge of the importance of these factors gradually becomes disseminated. With development of potent A_1, A_2, Rh and possibly other factors, it will be possible to increase the percentage of exclusions from the present approximately 40 per cent to a higher figure.

The use of the so-called "universal donor" (Group O or Type IV) may be distinctly dangerous if the recipient belongs to another blood group, because such a donor has in his serum the anti-A (α) and anti-B (β) agglutinins. Witebsky, Klendshoj and Swanson[22] set about the task of isolating the A and B specific substances and then adding them to blood containing anti-A and anti-B agglutinins, thus neutralizing the possible ill effects of transfusing Group O blood. Both the A and B substances are carbohydratelike and similar to the complex carbohydrates that are recognized as the bearers of the type specificity of pneumococci and other organisms. Addition of small amounts of these substances to blood for transfusions effectively removed the anti-A and anti-B agglutinins and did not cause reactions. These substances are not at present commercially available. They have been found of value not only in making Group O blood entirely compatible but also in neutralizing the A and B agglutinins of M, N and Rh testing serums.

The moral of this section on the blood groups might be stated as follows: transfuse only when absolutely necessary and only after extremely careful compatibility tests have been performed by responsible, experienced persons.

*These may be obtained from the Blood Betterment Association, New York City, and from Lederle Laboratories, Incorporated, Pearl River, New York.

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White Blood Cells

Basophils

Doan and Reinhart[23] present a carefully documented discussion of the basophil (basophil granulocyte). They state that "of all the cells found in normal blood least, perhaps, is known at the present time about the basophil type of granulocyte." They cite cases indicating a possible "primary progressive specific hyperplasia of basophil granule leukocytes."

Infectious Mononucleosis

This very common, and rarely diagnosed, condition continues to be the subject of numerous reports. In a paper by Werlin, Dolgopol and Stern,[24] the possible confusion of the condition with diphtheria is discussed. In 14 of 21 cases admitted to the Willard Parker Hospital in New York City, the clinical picture was considered sufficiently suspicious of diphtheria to warrant the administration of diphtheria antitoxin on admission. The authors state:

> . . . . The diagnostic difficulty was further complicated in 7 of these cases by the presence of non-toxic diphtheria-like bacilli in the throats of 6 patients and in the nose of 1 patient. The negative toxicity tests of the isolated bacilli, the hematologic picture, the positive heterophil antibody test and the lack of response to the diphtheria antitoxin helped in establishing the correct diagnosis.

Cerebral complications are quite frequent, and as Thelander and Shaw[25] point out, severe headache is often an early and prominent symptom. I have recently seen a number of cases in which the diagnosis of "aseptic lymphocytic meningitis" or "choriomeningitis" was made. To be sure, the spinal fluid revealed a lymphocytosis, but there was a marked lymphocytosis in the peripheral blood and the sheep-cell agglutination test was positive. Sadusk[26] directs attention to the temporarily positive serologic tests for syphilis, which occur in approximately 15 per cent of reported cases (only in some series, it should be noted) and indicate that a positive Wassermann or Kahn reaction is likelier to occur in the presence of a rash.

Smith[27] directs attention to what he calls "infectious lymphocytosis." In young patients, a transient lymphocytosis without other symptoms or signs, or a more prolonged lymphocytosis following infections of the upper respiratory tract, is not uncommon. Smith contends that these cases are frequently confused with infectious mononucleosis, but that they should be differentiated from them by the essentially normal, mature character of the lymphocytes, and by the negative heterophil reaction. This differentiation is significant, since otherwise an underlying respiratory or other infectious process might be missed. Essentially, Smith's infectious lymphocytosis is simply the lymphocytic reaction commonly seen in any chronic smoldering infectious process, and does not indicate a hitherto undescribed condition. What is more, infectious mononucleosis might best be called "infectious lymphocytosis," since the cellular reaction is primarily lymphocytic and not monocytic. Acute cases of so-called "glandular fever" that resemble mononucleosis in every respect but have negative heterophil reactions are occasionally seen. Are these due to a different etiologic agent than that causing infectious mononucleosis, or are they simply heterophil negative? Since the etiologic agent has not yet been discovered, this question is difficult to answer.

Leukocytic Reactions, including Leukopenia

The sulfonamide drugs continue to prove a fertile source for the development of various reactions involving both the red cells and the leukocytes, although with the use of the newer drugs, sulfathiazole and sulfadiazine, the reactions seem to be distinctly lessened. Rinkoff and Spring[28] report 8 cases of either severe leukopenia or agranulocytosis following the use of sulfanilamide or sulfapyridine, but of 51 treated with sulfathiazole, none developed a toxic reaction. However, Kennedy and Finland[29] report a case of fatal agranulocytosis following the use of sulfathiazole. Plum and Thomsen[30] compared the agranulocytosis caused by amidopyrine with that induced by the sulfonamides and concluded that an accompanying anemia was much commoner with the latter drugs and was often associated with a rash, although severe throat lesions were less frequent. The mortality rate was distinctly less in the sulfonamide-treated cases (67 per cent). Occasionally, marked leukocytosis and even a leukemoid reaction may occur with the sulfonamides. These are usually either in association with or preceding a definite hemolytic reaction. Whether or not some of the cases of acute leukemia seen following the use of sulfonamides have any relation to the drug or are coincidental cannot be stated for certain, although since the drugs are given for almost every fever, coincidence is more than likely.

Lawrence[31] describes the various abnormal physiologic conditions that may result in leukopenia, grouping them as follows: those due to diminished manufacture of white blood cells (including aplasia and maturation arrest); those due to increased elimination of white blood cells; those due to an increased rate of destruction in the peripheral blood; those due to redistribution of

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the leukocytes in the vascular channels; and those due to redistribution of the leukocytes in the body as a whole, as in the leukopenic phases of certain cases of leukemia. He makes a plea for a more physiologic understanding of the various causes of leukopenia. An interesting type of severe leukopenia said to be caused by an increased destruction of leukocytes by the spleen is described by Muether and his associates.[32] This condition, first described in a systematic way by Doan,[33] may be acute or chronic, and may even simulate agranulocytosis. It is often accompanied by splenomegaly, and at times by slight hemolytic jaundice. The bone marrow is hyperplastic. Splenectomy has resulted in dramatic cures. The interpretation of both the Columbus and the St. Louis workers is that the spleen destroys the leukocytes by phagocytosis. It is my belief[34] that splenic enlargement often causes an increase in the normal inhibitory effect of that organ in the marrow, with the result that the marrow becomes crowded with leukocytes, which are apparently prevented from being delivered to the circulating blood. Splenectomy may result in a resumption of the normal delivery mechanism. In any event, splenic leukopenia and granulocytopenia, often of striking degree, may be secondary to splenomegaly and relieved dramatically by splenectomy.

Reactions to Benzol and Related Chemicals

An increasing interest is being shown in the reactions of benzene chemicals on the blood-forming organs. Schwarz and Teleky[35] state that the blood picture in chronic industrial benzene poisoning is the result of a balance between destructive and regenerative forces. Instead of the classic anemia, leukopenia and thrombopenia, temporary erythrocytosis, erythroblastosis, leukocytosis with immature white cells and even leukemia may develop. Any change from the normal should be an immediate indication for the removal of the worker from the source of exposure. It is recommended that blood examinations be made monthly in workers using benzene, and every four to six months in those using related chemicals. Goldwater,[36] in a study of 332 workers exposed to benzol fumes in rotogravure industry in New York City, observed that anemia, macrocytosis and thrombocytopenia were the commonest abnormalities, whereas leukopenia and neutropenia were rather unusual. Not infrequently, relatively high hemoglobin values associated with elevations in the mean corpuscular volume were present. In another paper, Goldwater and Tewksbury[37] made a follow-up study of rotogravure workers who had developed blood changes as the result of exposure to benzol and who had then been safeguarded from the hazards. Toxic effects were present in some cases for at least two years. Recovery was manifested by an increase in red-cell count, a decrease in red-cell size and an increase in the platelet count. The usual forms of anti-anemic treatment appeared to have no effect in hastening recovery. Hamilton-Paterson[38] studied workers exposed to benzol in the rubber industry. In this series of cases, leukopenia was the commonest finding, but leukocytosis and even leukemia occasionally developed.

All these reports confirm an earlier one by Hunter[39] in which similar findings were made. The possibility that prolonged exposure to low concentrations of benzene might produce leukemia was first broached in this paper, and should be seriously considered. Careful histologic studies of the bone marrow and other blood-forming organs were made by Mallory, Gall and Brickley,[40] who showed the very variable changes — aplastic, hypoplastic, proliferative and even leukemic — that might develop. These changes depended on the intensity and duration of exposure, hyperplasia following prolonged exposure and hypoplasia of either short or long contact. The authors pointed out that although the evidence indicating that chronic exposure produces leukemia was still incomplete, it is nevertheless accumulating and worthy of serious consideration. Rawson, Parker and Jackson[41] state that all their 6 cases that were diagnosed as "agnogenic myeloid metaplasia" and were available for study gave a history of exposure to industrial solvents, "high-test" gasoline, carbon tetrachloride, paint remover or benzol.

Polycythemia Vera

Primary, idiopathic or true polycythemia is a disease of the bone marrow in which striking hyperplasia of all the elements — erythroblastic, leukocytic and megakaryocytic — continues for many years. This results in great increases in the red cells, leukocytes and platelets of the blood and, finally, in a great overdistention of the entire circulation. The cause of the continued hyperplasia remains obscure, although the most attractive theory is that of anoxia of the marrow, with resultant stimulation to erythropoiesis. It should be noted, however, that anoxia (such as that at high altitudes) causes only erythrocytosis, the platelets and granulocytes remaining normal in number. Davis[42] showed that the oral and subcutaneous administration of both ephedrine and amphetamine (Benzedrine) caused polycythemia (erythrocytosis) in dogs and rabbits, perhaps through vasoconstriction of the vessels of the bone marrow, with resultant anoxia and stimulation of erythropoiesis. This effect occurred in both

normal and splenectomized animals and appeared to be due to a real influence on red-cell production.

The extreme viscosity of the blood, in addition to the great overdistention of the entire circulation, leads to the development of a sluggishly moving mass of blood. Offhand, it seems that the work of the heart should be increased. However, measurements of the cardiac and respiratory function in 3 relatively young patients by Altschule, Volk and Henstell[43] revealed normal values, and the conclusion was made that the slowing of the blood flow was due to increased capillary resistance because of the greatly increased viscosity, which, with the slow blood flow, the greatly increased platelet count and the early onset of arteriosclerosis, is of course conducive to the development of thrombosis.

Another complication that has recently received increasing attention is that of sclerosis of the marrow usually associated with a bone-marrow type of cellular proliferation (myeloid metaplasia) in the spleen. In last year's progress report,[44] reference was made to a number of articles in which this end-result condition was discussed. That there is an "agnogenic myeloid metaplasia"[45] of the spleen, without reference to polycythemia, hemolytic anemia or involvement of the bone marrow by neoplasm, leukemia, Gaucher's disease and so forth, is questionable; nor does it seem desirable to make of this condition a separate entity. Furthermore, that bone-marrow proliferation develops in the spleen in response to bone-marrow destruction elsewhere or to unusual bone-marrow stimulation, as in severe hemolytic anemia, has been known for many years; its rediscovery under a new guise is interesting but perhaps confusing. It is also likely that many of the cases of chronic myelogenous leukemia reported following polycythemia are in reality examples of this "burnt-out" state of polycythemia, the leukocytic proliferation being unusually prominent.

In the treatment of polycythemia, all the symptoms may be quickly relieved by reducing the blood volume to normal and keeping it so. This is done by semiweekly venesections of blood for three to five weeks (depending on the case), and by the use of a diet low in iron.[46] On the face of it, this appears to be a more physiologic method of treatment than the use of hemolytic drugs (phenylhydrazine), a toxic drug such as lead, as suggested by Falconer,[47] or radioactive phosphorus.[48] Patients with polycythemia are relatively healthy and live for many years. The use of harmful or potentially harmful drugs seems therefore undesirable.

Hemorrhagic Diseases

The clotting of blood in blood vessels is a function of the enzyme-chemical factors, thromboplastin-prothrombin-fibrinogen, of the platelets and of the blood-vessel wall. Failure of clotting occurs in the presence of a defect in any of these three mechanisms. An analysis of 355 autopsy cases that showed one or another of these disturbances is reported by Perlman and Fox.[49] The suggestion is made that the term "hemorrhagic diathesis" be used, qualified by the phrase "on the basis of" damage to or weakness of the capillary endothelium, deficiency of the clot-forming elements or faulty clot retraction due to thrombocytopenia.

Deficiency of the Clot-Promoting Elements

Quick[50] gives an excellent and detailed review of the hemorrhagic disorders due to defects in the coagulation mechanism of the blood. These may be associated with diminished prothrombin, calcium deficiency, thromboplastin deficiency, fibrinogen deficiency and, finally, the presence of anticoagulants. Ferguson[51] also gives a summary of his and other work on the blood-clotting anomalies in the hemorrhagic diseases. Both authors rule out calcium as having any importance in these conditions. The original reviews should be consulted for specific information on these subjects.

Hypoprothrombinemia. The earlier work on vitamin K deficiency and its bearing on hypoprothrombinemia has been cited in previous reviews. Recent articles have extended the groundwork already laid. The value of protecting the newborn infant from the effects of a low prothrombin level by giving the expectant mother vitamin K from five to twenty-eight days prior to delivery is pointed out by Valentine, Reinhold and Schneider.[52] About three quarters of the infants of untreated mothers developed a low prothrombin level within forty-eight to ninety-six hours after birth, whereas infants of protected mothers showed no such fall. It is further to be noted that one of the causes of cerebral hemorrhage of the newborn may be hypoprothrombinemia, and that the prothrombin level of the infant may be made normal by the simple injection of one of the parenteral preparations six or eight hours before delivery. That liver damage results in a diminution in the prothrombin is by now well known. Lucia and Aggeler[53] tried to correlate the various liver-function tests with the diminished blood prothrombin in a series of cases. No definite correlation could be determined, although some differentiation between hepatic jaundice and obstructive jaundice could be worked out by the administration of vitamin K. In the presence of hepatic disease, the prothrom-

bin clotting time could not be materially altered. Similar findings and the development of a liver-function test based on this procedure are made by Lord and Andrus.[54] A hemorrhagic agent resulting in an increased prothrombin time has been studied by Bingham, Meyer and Pohle.[55] This material, which was isolated and crystallized at the University of Wisconsin Agricultural Experiment Station, has been shown to be a dicoumouran. Synthetically prepared, it produced prolongation of the prothrombin and coagulation times, when given to dogs and human subjects. It is suggested that the material may prove to have a valuable place in clinical medicine as an anticoagulant to replace heparin.

Hemophilia. Despite vigorous investigation, no very striking advance has occurred in either the pathogenesis or the treatment of hemophilia. The researches of Taylor and his collaborators at the Thorndike Memorial Laboratory, Boston City Hospital, cited in previous reviews, indicated that something was lacking in the globulin fraction of the plasma. In the meantime, the old theory that hemophilia is due to a diminution in thromboplastin formation from unusually resistant platelets has been espoused again, chiefly by Quick,[56] Ferguson[51] and Howell.[57] Quick described a new diagnostic test for hemophilia based on the diminished production of thromboplastin from abnormally resistant platelets. He found that the coagulation time of oxalated hemophilic plasma subjected to high centrifugation is markedly longer than that of slowly centrifuged plasma. With high centrifugation, the intact platelets are thrown down, leaving a plasma poor in both free and potentially free thromboplastin. Ferguson suggests that hemophilia is a deficiency in the plasma content of a new thromboplastic enzyme, the function of which is to mobilize calcium and cephalin for the conversion of prothrombin to thrombin. Howell considers one real difference between normal and hemophilic blood to be that the latter contains less thromboplastin in its plasma.*

Although most of the complications of hemophilia cannot be controlled except by the transfusion of fresh blood or plasma, an advance has recently been made in the control of bleeding from a local exposed area, such as a tooth socket. This has come out of the investigations of Parfentjev,[58] who by purification of the globulin fraction of rabbit plasmas obtained a substance capable of producing clotting when added in very small amounts to normal plasma or blood. This material was found to act directly on fibrinogen and could clot prothrombin-free plasma. Parfentjev's results were confirmed by Taylor, Lozner and Adams,[59] who demonstrated that the material was a pseudoglobulin possessing true thrombic activity and differing from thromboplastin. It could be prepared in dry, stable form. Lozner, MacDonald, Finland and Taylor[60] found that one part of dry powdered rabbit thrombin could clot 60,000 parts of 0.25 per cent citrated blood within three seconds, and was of distinct value in the control of hemorrhage from small wounds both in normal subjects and in those with various hemorrhagic diatheses, including hemophilia. It is probable that this material will shortly be commercially available.

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Although heparin, which is an anticoagulant, is somewhat outside the scope of this review, one should mention the monograph on this subject by Jorpes[61] and a recent symposium[62] on heparin and thrombosis. Ferguson and Glazko[63] have also contributed a short review.

Thrombocytopenic Purpura

The controversy still continues about whether the bleeding in thrombocytopenic purpura is due solely to a deficiency in the blood platelets or to a vascular factor. MacFarlane,[64] in a comprehensive and critical review of hemostasis, concludes that the platelets, aside from their role as accelerators of blood coagulation and clot retraction, do not have an essential part in the hemostatic mechanism, but that the contractility of the blood-vessel wall is all important. In idiopathic thrombocytopenic purpura, the significance of the platelet count is denied, but the capillaries of the nail bed are very irregular and distorted. These observations hardly agree with the almost instantaneous response to splenectomy and the striking changes of the megakaryocytes in the marrow before and after the operation. Rabinowitz[65] contributes yet another theory, namely, that the liver function in the disease is somehow impaired. The oral administration of an amino-acid, methionine, is stated to be effective in controlling the spontaneous bleeding and the defective clot retraction, and to be "of inestimable value in the management of both the acute and chronic stages of the disease." In view of the many substances that have been recommended in the treatment of thrombopenic purpura, the results with methionine must await confirmation. Elliott and Whipple[66] conclude that the role of the spleen in the disease is active and important, although the capillary factor is also of consequence. It should be remembered, however, that the pathognomonic feature of idiopathic thrombopenic purpura is the great reduction in the blood platelets, and that the disease is almost always cured, capillaries or no capillaries, by

*The work of these three investigators contradicts that of Taylor and his associates, which indicates that platelets have little, if any, relation to the clot-promoting globulin substance.

splenectomy, which results in a dramatic and sustained rise in the platelet count.

The exact relation of the spleen to the disorder is not entirely clear, although it is my contention that some sort of functional abnormality develops there and causes an unusual inhibition of platelet formation from megakaryocytes in the bone marrow. This is based chiefly on bone-marrow studies before and after splenectomy. Another method of approach is that of the injection of splenic extracts prepared from splenectomized cases. Troland and Lee[67] reported a remarkable reduction in the platelet count of rabbits when an active extract ("thrombocytopen") was injected. A number of investigators have failed to confirm this apparently clear-cut finding; on the other hand, Rose and Boyer[68] and, more recently, Otenasek and Lee[69] offer complete confirmation in several cases.

What is clear in the disease is the dramatic effect of splenectomy. With a patient bleeding acutely from all the orifices and into the skin, it is first essential to rule out acute leukemia or other disease and at the same time rule in the idiopathic form of thrombopenic purpura. The blood picture does not always permit this differentiation, and for this reason, it is my custom always to perform a sternal puncture. If the marrow shows no evidence of leukemic or other proliferative lesion but presents large numbers of megakaryocytes without platelet formation, immediate splenectomy is recommended, because if the natural procrastination is allowed to win out, a fatal hemorrhage into a vital organ — usually the brain — may well occur. In the past year, I have seen 3 such cases, in which for various reasons the decision for splenectomy was delayed; in the meantime, while the discussion was continuing, the patient in each case had a cerebral hemorrhage and died. In contrast to this, in a recent dramatic case, a young woman had an appendectomy and almost simultaneously began to bleed into the abdominal wound and the skin, and from all the orifices. Despite several transfusions, her condition soon became desperate. There was marked anemia and tachycardia. After study of the blood and marrow indicated an idiopathic thrombopenic purpura, immediate splenectomy was recommended despite the obvious surgical risk and the recent abdominal operation. Splenectomy resulted in quick cure. In this case, the almost 100 per cent mortality with watchful waiting was balanced against the perhaps 30 to 50 per cent mortality of splenectomy.

Vascular Purpura

Bleeding into the skin or mucous membranes may take place in the presence of normal platelets and under conditions of capillary damage induced by infectious, toxic, allergic or unknown agents. The "devil's pinches" so common in women and the purpura of nephritis and of the Schönlein–Henoch syndrome are of this vascular type. A deficiency in ascorbic acid leads to the capillary hemorrhages so common in scurvy. Vitamin C has been tried for many types of vascular purpura, but it is completely without effect except when a deficiency in that vitamin is present. Recently, vitamin P or citrin (Szent-Györgi) derived from lemon juice or orange peel has been suggested as a regulator of vascular permeability and, hence, as an effective agent in the treatment of vascular purpuras. Kugelmass[70] reported good results with this material in the treatment of 2 cases of allergic purpura, 1 of infectious purpura and 1 of nutritional purpura. In a case of purpura hemorrhagica occurring after arsenic therapy, Gorrie[71] reported quick recovery following treatment with vitamin P (Hesperidin, Glaxo). These results deserve further study before their real value is determined.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 28101

PRESENTATION OF CASE*

A twenty-three-month-old Portuguese-American girl was admitted to the hospital because of dark-colored urine.

One week before entry, she began to void dark urine and to pass light-colored stools. For two days previous to entry, she was feverish, and it was noted that the abdomen was enlarged. She had no vomiting, diarrhea or other systemic or constitutional symptoms.

The family history was not contributory. The patient was a full-term, normally delivered infant who had received an adequate diet and had undergone normal growth and development.

Physical examination revealed a well-developed and well-nourished child whose breathing was rapid and shallow, but regular. There was moderate icterus of the skin and scleras. Shotty lymph nodes were palpable bilaterally in the cervical and inguinal regions. The heart and lungs were normal. The abdomen was protuberant and tense. The liver edge was palpable at the level of the umbilicus. There was no spasm or tenderness. The spleen and kidneys were not palpable, and no fluid wave was obtained. Examination of the nervous system was negative.

The temperature was 99.6°F., the pulse 140, and the respirations 28.

Several samples of urine were deep brown, with moderate amounts of bile. There were slight traces of albumin on several occasions, with repeatedly negative sediments. The urine was free of urobilinogen. Examination of the blood showed a red-cell count varying between 3,560,000 and 5,100,000 with corresponding hemoglobin determinations between 69 and 83 per cent, and white-cell counts ranging between 13,000 and 19,000 with a normal differential. The blood Hinton reaction was negative. Repeated blood cultures were negative. The venous clotting time was 7 minutes, with a normal clot retraction. The fasting blood sugar was 182 mg. per 100 cc., and the blood cholesterol 268 mg. The van den Bergh reaction was biphasic, and the nonprotein nitrogen of the blood serum was 28.7 mg. per 100 cc. The stools were constantly clay colored. Repeated brucellergin tests and tuberculin tests in dilutions up to 1:10 were negative. Investigation of the sputum for fungi was negative.

Roentgenograms of the skull and extremities were negative, and those of the lungs showed a moderate amount of diffuse, fine, mottled peribronchial infiltration. On repeated examinations of the chest, the infiltration became more diffuse over a two-week period and then remained unchanged until three months later, when the lung fields became more vesicular and emphysematous.

The patient remained in the hospital for four months. During this time, she ran an intermittent low-grade temperature, with occasional bouts of fever up to 103°F. Her condition remained essentially unchanged except for a slight but steadily progressive loss of weight. Jaundice persisted. Two and a half weeks after entry, the spleen became palpable. Three and a half months after entry, a lymph-node biopsy was interpreted as showing chronic nonspecific lymphadenitis. Four months after admission, an exploratory laparotomy was performed. Although the patient apparently withstood the procedure quite well, death occurred on the first postoperative day.

DIFFERENTIAL DIAGNOSIS

DR. HARRY L. MUELLER: We might pause for a moment to remark that this is a Portuguese infant and that the onset of the jaundice may therefore have been at an earlier date than was suspected, because these children are rather apt to have dark, olive skins.

This patient was in the hospital for four months. If she had a progressive constitutional disease, it is surprising that her condition did not become much worse during that period than is apparent from this history.

Painless, persistent jaundice, with clay-colored stools, and the large liver persisting for four months in a two-year-old infant certainly make me believe that there was some unusual cause of obstruction to the biliary drainage system. The fact that the child exhibited no gastrointestinal symptoms at an age when almost any disease is accompanied by such symptoms makes me quite confident that there was no disease in the gastrointestinal tract. I am ruling out catarrhal jaundice, which is the common cause of jaundice in a child, although quite rare at this age, because of the long course and the fact that there were no gastrointestinal symptoms.

We must assume a point of obstruction and select

*This case is presented through the courtesy of the Children's Hospital, Boston.

the most likely point and cause for that obstruction. I should consider the portal fissure the most probable position, and it appeals to me to place Hodgkin's disease in that region. The loss of weight, fever, pulmonary lesion and apparently normal blood picture, as well as the later development of splenic enlargement, are all consistent with Hodgkin's disease. This condition may present an apparently normal blood picture, occasionally has a leukocytosis, and may have a relative lymphocytosis, which might explain why the differential count appeared normal, although there was a leukocytosis. The negative biopsy does not to my mind rule out Hodgkin's disease. Another lesion that should be given serious consideration is lymphosarcoma in the same region, although the course of the disease does not fit in so well. Lymphosarcoma is prone to metastasize to both the bronchial and the mediastinal lymph nodes, and such metastasis must be considered. Tuberculosis in this position does not often cause pressure symptoms, and with a negative tuberculin reaction and the lack of any report of calcification I should be inclined to rule that out. We might stop here and get an interpretation of the x-ray films.

DR. AUBREY O. HAMPTON: This is a very unusual pulmonary picture. In the first examination, it looks very much like miliary tuberculosis. In fact, that would be my unhesitating opinion if I saw this film alone. I should think that it was in the terminal stage and that the patient would die much sooner than in three months — in fact, three days would be more likely. However, the pulmonary picture changes markedly in three months and shifts into something entirely different. At first, there was a miliary process in the lung, and at the last examination there is what appears to be pulmonary fibrosis with emphysema. I have not seen pulmonary fibrosis that started out as a miliary lesion. In some respects, the progression is like silicosis, which can begin with a miliary lesion, and later shifts into extreme fibrosis with blebs, but a child does not have silicosis. The marked increase in the anteroposterior diameter of the chest is like emphysema. The abdomen is rather unusual. Here is the shadow of what appears to be the liver. It is unusually distinct in the lateral view; it is not so distinct in the anteroposterior view, but it is certainly enlarged. I think that the spleen is represented here — moderately enlarged, not markedly. That film was taken four months before death.

DR. MUELLER: Can you make out any kidney shadows?

DR. HAMPTON: The right kidney appears normal in size and shape. I do not see the left.

DR. MUELLER: Is there any question of a retroperitoneal mass?

DR. HAMPTON: No; and I do not believe that lymphoma would produce this chest picture unless a great deal of radiation had been given.

DR. MUELLER: Could Hodgkin's disease regress as the result of frequent x-ray and fluoroscopic studies?

DR. HAMPTON: This is a very diffuse lesion. All the lobes are involved alike. I do not believe we could produce that picture with radiation if we tried.

DR. MUELLER: Is there any evidence of enlargement or calcification of the mediastinal lymph nodes?

DR. HAMPTON: No.

DR. MUELLER: I do not know much more than I did before we started except that I am a little more confused. I am convinced that this child had an obstructing lesion somewhere in the biliary drainage system. We must try to couple it with the pulmonary process and decide whether the primary lesion was in the lungs or in the abdomen.

Malignant tumors in infants of this age have a very rapid course, and I do not believe that the child would have been in such good condition at the end of four months if the metastases had been so widely distributed. There is also no evidence of a palpable mass, although that could be tucked up under the edge of the liver. Stone seems very unlikely, with the other aspects of the case, and of course is rare in a child of this age. I do not believe we have to consider it seriously. Foreign body should always be considered in children. Any foreign body that would obstruct the biliary tract would certainly cause some gastrointestinal symptoms. Parasites should perhaps be more seriously considered than I had thought previously. Distoma and ascaris can both invade the common and intrahepatic ducts, as well as the cystic, and can cause obstruction. I should expect stool examinations to pick up some evidence of that. There is nothing in the record to show that the stool examinations were made with that in mind. One other parasite that ought to be considered is the hydatid cyst. The Portuguese are sheep herders. There is no mention in the record that these people had a sheep dog, but a hydatid cyst impinging on the hepatic duct could give a course similar to this. What type of lesion might result in the lungs from ruptured hydatid cyst, I am frank to say I do not know, but I do not believe that has to be considered seriously. Tumors of the pancreas and gastrointestinal tract and kidney are rather unlikely because they would present other gastrointestinal or urinary signs and symp-

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toms, none of which were present in the entire course. Wilms's tumor must always be considered in a child and on the right side can cause obstruction of the biliary tract. It metastasizes early, and I doubt whether it can present such a picture as this in the lungs. It also metastasizes to bones, and x-ray films of the long bones were negative. Primary tumor of the liver itself seems unlikely. The liver was smooth; it showed no irregularities, and these tumors grow very rapidly and I believe would present other evidence of cancer in the patient.

Turning to the question of infection, syphilis should be considered. Against it are the negative blood Hinton reaction and the absence of changes in the long bone and of other stigmas. Brucella and fungous infections, which have been quite well ruled out by the clinicians, would not present persistent obstructive jaundice with clay-colored stools for that period. Liver abscess is, I think, a greater possibility than I originally considered. It possibly could be the source of multiple septic foci in the lung. It would be unusual for such foci to become fibrosed as these did, but this is an unusual case and we must consider every possibility. Abscess can run a fairly silent course in the liver itself and be very difficult to diagnose and sometimes, is diagnosed only by its metastatic lesions. Congenital anomalies are always a source of worry to the pediatrician. Liver cysts, cysts of the biliary ducts and stenosis of the ducts could cause half this picture but not, I believe, the complicated picture in the lung.

I come back to my original thought that, to make a diagnosis in this case, an exploratory laparotomy would probably have been necessary, and whether it was done for that reason I do not know. I should say that this child had obstructive jaundice. I do not know to what the lesions in the lungs are due. I had picked as my first choice Hodgkin's disease, but with this picture in the lungs I tend more to favor infection and should say liver abscess or localized infection in the portal fissure. I should also like to put in a guess, because of the patient's nationality, that there is a possibility of a hydatid cyst.

DR. TRACY B. MALLORY: Dr. Hampton, would you be willing to make a diagnosis?

DR. HAMPTON: No; I have not the vaguest idea. I should like to know whether an oil spray was used. It could produce that picture in the lungs.

CLINICAL DIAGNOSES

Biliary cirrhosis, obstructive.
Pulmonary fibrosis, chronic, nonspecific.

DR. MUELLER'S DIAGNOSES

Obstructive jaundice.
Liver abscess?
Hodgkin's disease?
Hydatid cyst?

ANATOMICAL DIAGNOSIS

Hand–Schueller–Christian syndrome, with involvement of liver, lungs, lymph nodes, bone and so forth causing interstitial emphysema (marked) and obstructive biliary cirrhosis.

PATHOLOGICAL DISCUSSION

DR. SIDNEY FARBER*: The clinical diagnoses were obstructive biliary cirrhosis and chronic, nonspecific pulmonary fibrosis. The exact nature of the disease was not understood by the clinicians during the life of the child, and nothing was learned from the clinical studies. As Dr. Mueller pointed out in the clinical history, the child was not malnourished and was really more unhappy than desperately ill throughout most of the course.

At autopsy, a granulomatous process that involved many parts of the body but mainly the lungs, the lymph nodes, the liver and the bone marrow, and also the kidney, uterus and tongue, was found. There was an obstructive biliary cirrhosis secondary in part to pressure on the common duct caused by enlarged lymph nodes and a mass of granulation tissue, and in part to compression of the intrahepatic bile ducts by masses of granulation tissue and granulomatous involvement of the liver parenchyma. A considerable amount of fibrous-tissue replacement of destroyed liver parenchyma was present throughout the liver. The greater amount of obstruction was caused by pressure on the common duct. The extraordinary x-ray picture of the lungs was caused by diffuse destruction of the alveolar walls and infiltration of the interstitial tissues of the lungs by the same type of granulomatous process observed in the extrahepatic lymph nodes and in the liver. Secondary to the destruction of alveolar walls, an escape of air into the interstices of the lung occurred, causing numerous small blebs and pseudocysts widely scattered throughout all lobes of both lungs. Histologically, the granulomatous process in the various parts of the body was characterized by the presence of large mononuclear cells, the cytoplasm containing large or small droplets of lipid or cellular debris, varying degrees of fibrosis and a cellular reaction composed of lymphocytes, occasional plasma cells and, in rare places where necrosis was present, polymorphonu-

*Assistant professor of pathology, Harvard Medical School; pathologist, Children's Hospital.

clear leukocytes. The lipid material in the large mononuclear cells stained readily by the scharlach R method, and some of the lipid was definitely doubly refractile.

The exact cause of this granulomatous process is unknown, and no further evidence of etiologic value was obtained from this post-mortem examination. Material from the lung and from the liver was injected by a variety of methods into a number of laboratory animals without the production of any lesion. Bacteriologic study of the heart's blood and tissue from the liver and spleen by aerobic, anaerobic and partial tension methods revealed no growth. The gross and histologic findings were consistent with what has been described as one variant of the Hand-Schueller-Christian disease, and what some writers have called Letterer-Siwe disease. Some patients with lung changes of the same type as those described here have had, in addition, numerous small skin lesions, which are often taken for tuberculides. Some of these patients have had destructive lesions of the skull or other parts of the skeleton. The clinical picture is often obscured by the peculiar localization of the pathologic process. In this patient, the icterus was the most significant part of the clinical picture and is explained mainly by accidental obstruction to the outflow of bile by involvement of the extrahepatic lymph nodes. Some patients have severe destruction of the petrous portion of the temporal bone. After operation, a foul purulent exudate, which may persist for weeks or months, may be found, bacterial infection being of only secondary importance.

A study of material obtained at operation and at autopsy of patients of the type under discussion and from patients with less dramatic visceral involvement, or with lesions limited apparently to the skeleton, has forced me to adopt a working hypothesis that Hand-Schueller-Christian disease, Letterer-Siwe disease and the condition that has come to be known in the last year or two as eosinophilic granuloma of bone or solitary granuloma of bone are all variants of the same disease. Certainly, it is generally recognized that the classical Hand-Schueller-Christian triad of exophthalmos, diabetes insipidus and skeletal involvement represents but one manifestation of the basic pathologic process. The disease appears to be a self-limiting granuloma. Of greatest consequence in the prognosis is the anatomic localization of the lesions.

A PHYSICIAN: Are the lesions radiosensitive?

DR. FARBER: Yes; we have not had very much experience, but we have learned that the bone lesions particularly are extraordinarily radiosensitive. At this time we have a patient on the wards of the Children's Hospital with liver involvement similar to that of the patient under discussion today. The liver is receiving radiation therapy.

A PHYSICIAN: Why did they operate?

DR. FARBER: The operation was performed in part to relieve biliary obstruction, if possible, and in part to find out the nature of the pathologic process.

FRANKLIN SQUARE HOSPITAL

CASE 28102

PRESENTATION OF CASE

An eleven-year-old schoolboy, apparently in good health, while playing vigorously outdoors suddenly fell unconscious, took a few breaths and died. A slight sanguineous oozing appeared from the mouth.

At birth, the patient had been delivered at term by forceps but appeared to be well. During his first year, he had presented a difficult feeding problem. At the age of two years, he had a moderately severe upper respiratory infection; at four, he had an attack of unexplained high fever for a few days culminating in a convulsion and recovery.

At five years of age, he had a severe illness that began as a bowel upset with eight to nine movements a day and a little mucus in the stools. The family physician noted for the first time slight cardiac enlargement, a rather rapid rate and a slight apical systolic murmur. There was no fever, rash, epistaxis or joint pains. In two or three days, the bowel irregularity subsided, but the heart action continued overactive without obvious cause. The patient remained quietly at home. Two weeks later, he awoke at midnight complaining of abdominal pain. He was taken to another hospital, where he was found to have a normal temperature. He was pale but not cyanotic. The respirations were rapid. There was considerable puffiness of the face and hands, and slight pitting of the lower legs. The throat was normal, and the lungs were clear. The cervical veins were swollen and pulsating, and the liver was large and very tender and extended to the umbilicus.

The heart was considerably enlarged, rapid and regular at 140. The most striking auscultatory finding was a marked gallop rhythm. There was also a soft apical systolic murmur, which later was inconstant from day to day and ultimately disappeared.

The urine was normal on several examinations. The blood showed a red-cell count of 4,700,000 with a hemoglobin of 94 per cent, and a white-cell count of 7600 with a normal differential count and smear.

An electrocardiogram showed a sinus tachycardia at 130 to 140, and moderate left-axis deviation. The T waves were low in all leads and flat in Lead 1. The P waves were rather high. The PR interval measured 0.17 second.

An x-ray film showed generalized enlargement of the heart both to the right and to the left, without characteristic shape. The transverse diameter was 11.3 cm., and the internal diameter of the chest 18.8 cm. The lungs were normal.

In the hospital, the patient was given 5 gr. of digitalis the first day, 8 gr. the second day, and 4½ gr. the third day, resulting in a striking slowing of the rate from 140 to 100 and a consequent marked improvement in the clinical picture — namely, a brisk diuresis, improvement in the quality of the heart sounds and a lessening in the intensity of the gallop to the order of a moderately loud third sound. No murmur was then audible. The peripheral edema disappeared in three or four days, and after ten days the liver receded to the costal margin. Two later x-ray films showed no significant change in heart size, and three subsequent electrocardiograms showed slightly less left-axis deviation than that on admission but otherwise remained unchanged, including the PR interval. The patient was discharged after a month of hospitalization on a daily ration of ¾ gr. of digitalis.

During the next six years, up to his sudden death, the patient remained in good health, grew normally, and was precocious mentally. His activities were essentially normal for a boy of his age. He was followed by his physicians about twice a year, and at times there was a tendency to mild tachycardia. On these occasions, the gallop rhythm reappeared but subsided with rest and slight increases in the digitalis ration. The heart remained enlarged.

Two years before death, — because of vague abdominal discomfort, — a gastrointestinal series was done in this hospital. The first portion of the duodenum showed a redundant loop crossed by a pressure defect. This deformity, which could be obliterated by manipulation, suggested to the roentgenologist an anatomic variation of the duodenum caused by an aberrant mesenteric artery.

One month before death, at the time of a routine checkup, the patient was well except for a slight respiratory infection. There was no cyanosis or clubbing and no palpable liver or spleen. There was a slight prominence of the left chest anteriorly. The cardiac apex was visible in the fifth interspace 8 cm. from the midsternal line and 2 cm. outside the midclavicular line. The action was quiet and regular at 80. There were no murmurs. In the recumbent position, there was a slight third sound. The blood pressure was 95 systolic, 65 diastolic. Under the fluoroscope, the heart was moderately enlarged and globular in shape. The transverse diameter was 11.6 cm., and the internal diameter of the chest 19.5 cm. The lungs were normal. An electrocardiogram showed moderate to marked left-axis deviation and a PR interval of 0.20 second. The T waves in Lead 1 were inverted, and the P waves were slightly prominent. It was thought that the patient was doing well.

Differential Diagnosis

Dr. Paul D. White: If this eleven-year-old boy had heart disease, — and his end sounds like a cardiac death, since most cerebral deaths are not so speedy, — the kind should be, in the order of probability, rheumatic, congenital or (quite possibly in the present case) something else. If it was either rheumatic or congenital, it was a rare case.

I cannot make much of these first events in the past history in view of what followed. Rheumatic fever generally does not take the course of a high fever culminating in a convulsion.

Almost any illness at the age of five may, however, excite acute active rheumatic heart disease, and it is possible that the sickness at that time induced the first manifestation of rheumatic or rheumatic-like heart disease with cardiac enlargement and rapid heart rate. From the intestinal trouble, I tried to work in some parasitic myocardial disease beginning during this illness, but I was unable to do so.

We do not know just how severe the abdominal pain was, except that it was severe enough to wake the patient up. One wonders if it was a complication associated with heart failure, such as liver engorgement. Pericarditis, dry or with effusion, sometimes gives rise to epigastric pain.

The patient had been flat in bed, so that this generalized edema may have been either cardiac or renal. So far as the cardiac examination is concerned, the edema might have been secondary to pericardial effusion, or it might more likely have been due to congestive heart failure. One hears a gallop rhythm frequently in constrictive pericarditis, but not in cases of acute pericarditis with effusion. The gallop rhythm is, however, much more in favor of cardiac dilatation. It can be induced by heart block alone, with long PR intervals in the electrocardiogram; the auricular contraction can then be heard.

Was the blood pressure noted at that time?

Dr. Edward F. Bland: It was essentially the same as it was later on.

DR. WHITE: The fact that the urine was normal on several occasions is important. We do know that at this age an acute nephritis may be associated, as Dr. Longcope and others have shown, with cardiac dilatation and often with failure that may take months to clear up.

The left-axis deviation is suggestive of something unusual at five, for even when of moderate degree it is rare at this age. The low T waves were abnormal and fit in with pericardial or myocardial disease, leaving out, of course, a state of myxedema or some other rare factor that would be very unlikely in this boy. The PR interval was rather long, but not excessively so.

Are there any x-ray films?

DR. AUBREY O. HAMPTON: This film was taken six years before death. There was not much change in later examinations.

DR. WHITE: This film shows generalized enlargement of the heart. The shadow is rather globular and not diagnostic, so far as I know, of any one condition. There is, of course, so-called "idiopathic cardiac hypertrophy," congenital or not, in young children, which usually does not allow survival to this age. The x-ray picture is sometimes like this one. Such cases are, however, being subdivided, and an ample cause for the hypertrophy is usually found.

DR. HAMPTON: Do you want to explain why the left main bronchus is elevated? I think it is an unusual finding for almost any type of heart disease unaccompanied by left auricular enlargement.

DR. WHITE: Enlargement of the left auricle is certainly the usual cause for such a blunt-angled bifurcation of the trachea, and yet there is no particular reason to think that the left auricle was predominantly enlarged in the present case. I do not know that I have seen it in congenital enlargement of the heart. We have, however, no oblique view to indicate the presence or absence of some enlargement of the left auricle.

The patient was able to tolerate a good deal of digitalis — most children can. The result was an excellent classic digitalis effect in the presence of normal rhythm. Undoubtedly, the digitalis was responsible for the improvement in this case. He apparently continued with digitalis throughout the rest of his life. Is that true, Dr. Bland?

DR. BLAND: That is correct.

DR. WHITE: "No murmur was then audible." This is unusual in the presence of rheumatic heart disease because even dilatation, which may persist, results in most cases in a permanent murmur or murmurs, whether the valves are deformed or not.

The patient left the hospital, to remain away for six years, in a fair state of health, except for one interval. He was, however, really a patient for those six years, not a hale and hearty, strong boy. The myocardial reserve seems to have been distinctly limited.

The aberrant mesenteric artery is very intriguing. It may be a clue to anomalies elsewhere.

The absence of cyanosis and clubbing rules out quite definitely the cyanotic type of congenital heart disease. Rarely, the tetralogy of Fallot may be associated with only slight cyanosis, but I do not believe that we can consider the usual causes for the cyanotic type of congenital heart disease in this case.

The slight prominence of the left chest anteriorly is due, I suppose, to the marked enlargement of the heart during growth, which forced the chest wall forward.

Did the fluoroscopic examination show the same findings as the earlier x-ray film?

DR. HAMPTON: Yes; both recorded a transverse diameter of about 11 cm.

DR. WHITE: The heart had not grown rapidly larger. That is, it stayed large without great change all those six years, while the body was growing. Digitalis may have helped to maintain fairly good myocardial tone and thus may have prevented greater enlargement.

This electrocardiogram might be the record of a very large heart, with hypertrophy predominately of the left ventricle. Inversion of T waves does not necessarily mean coronary-artery disease. A large left ventricle, with or without coronary disease, may produce this picture.

The death sounds like that due to coronary insufficiency. As I said in the beginning, the diagnosis must be heart disease of either rheumatic, congenital or unknown cause, and so far as I can make any positive diagnosis, it must be labeled "unknown." But I shall make an attempt. It is obvious that the patient had a large heart, particularly involving the left ventricle. There is no reason to doubt that. Whether the left auricle was large and what the other heart chambers may have shown, I do not know. I suspect that the heart was enlarged throughout, though in the left ventricle predominately.

There was no evidence of hypertension, either systemic or pulmonary, and no evidence of valvular defect. There was myocardial disease, I think, of some sort — what kind, I cannot determine. Was it rheumatic without valvular disease? I wonder if enlarged hearts of unknown origin that we encounter in rare cases in adult life may

not be rheumatic after all, even in the absence of a positive rheumatic history or of any valvular deformity, left over from an insidious rheumatic infection involving the myocardium alone in childhood. There probably are a few of these cases, but of course we cannot prove them. And they are not common. Or this child's heart disease may have come directly from the sudden severe and obscure intestinal illness; we are, however, not acquainted with that kind of heart disease, if it exists. The patient had not had diphtheria. The electrocardiogram did not resemble such an effect. Was it toxic poisoning of unknown nature? Or was it a congenital anomaly of the blood supply? I am intrigued by that idea. This patient is rather old for it, and yet in France during the last war, in 1916, I attended the autopsy of a young soldier with an anomalous coronary circulation. His death was sudden, and we thought that it was probably due to an insufficient coronary blood supply. Consequently one can live to the age of eleven and beyond and die suddenly with an anomalous coronary blood supply. In the very striking case that we had in this hospital several years ago, the patient did not survive longer than a few months after birth. That patient had a left coronary artery that arose from the pulmonary artery and supplied the left ventricle with venous blood. We thought that, despite his age, he had angina pectoris. The electrocardiogram showed inversion of the T waves in Lead 1. Later, we found the congenital anomaly and a very large heart with extensive myocardial degeneration throughout the left ventricle. Perhaps the aberrant mesenteric artery is a clue to anomalous circulation elsewhere. Could this have been von Gierke's, that is, glycogen-storage, disease? The boy was distinctly old for it, but I do not know what the oldest case on record is. The lesion might, I suppose, have been rhabdomyoma, which I know very little about except that most patients do not survive nearly to this age; most of them die within a few months after birth.

I shall guess one of the bizarre diagnoses this time; perhaps a more likely one might be rheumatic or unknown heart disease, but I favor a congenital anomaly and put my first bet on anomalous coronary blood supply and my next on von Gierke's disease; a remote possibility is some tumor such as a rhabdomyoma. My second choice as to type is rheumatic heart disease of very unusual form, and my last choice is something else that may well have been the answer that I may not even have mentioned.

Dr. Bland: I first saw this boy during his severe illness six years ago at the Children's Hospital at the request of the late Dr. Kenneth Blackfan. We were much impressed by his rapid improvement on digitalis; so much so that we accepted it as clinical evidence against a serious myocarditis on an infectious basis, such as rheumatic, diphtheritic or secondary to acute nephritis. Thereafter, the patient lived a fairly normal and very active life. He was closely followed because of our special interest in what we considered a most unusual form of heart disease and also because of his parents' natural apprehension about his future.

The only other comment I should like to make is that I was intrigued by Dr. Hampton's suggestion two years before the patient died that he had an aberrant mesenteric artery, because it gave some support to Dr. Blackfan's and my opinion that this was a congenital idiopathic hypertrophy of the heart. I do not know the exact explanation for the unexpected death.

Dr. Howard B. Sprague: I suppose that the patient might have had a three-chambered heart. The electrocardiogram is not like that in our series of one case, but it is a possibility.

Clinical Diagnoses

Congenital idiopathic hypertrophy of the heart.
Aberrant vessel in region of duodenum.

Dr. White's Diagnoses

Marked cardiac enlargement, of unknown cause.
Congenital anomaly of the coronary circulation?
Von Gierke's (glycogen-storage) disease??
Rheumatic heart disease, without valvular deformity???

Anatomical Diagnosis

Hypertrophy of the heart, cause unknown.

Pathological Discussion

Dr. Tracy B. Mallory: This is the type of case in which the Pathology Department has to confess complete failure too. This patient had a very large heart — 240 gm.; the estimated normal size for a boy of his age would be around 112 gm. This was twice the normal size. All the chambers of the heart took part in the dilatation and hypertrophy quite uniformly, with no evidence of preponderance in either side. Besides hypertrophy, the only definite finding was a generalized thickening of the endocardium, notable in both ventricles and even a little more marked in the left auricle. The mitral valve showed two or three spots of fibrous thickening that did not suggest rheumatic endocarditis. The chordae tendineae were not in the least thickened or short, and there was no interadherence of the leaflets. Microscopically, there was no valvulitis. We cut a great many sections of different portions of the

myocardium, and there was no fibrosis and nothing to suggest an Aschoff nodule or any arterial involvement. The coronary arteries were normal in size and location. The aberrant artery, which Dr. Hampton suggested, was found crossing the duodenum. There was an azygos lobe of the lung. Possibly, these two things could be interpreted as evidence of congenital defect, and we can call this congenital hypertrophy. I know no way of settling the matter. I can simply make a diagnosis of hypertrophy of the heart, cause unknown.

Dr. White: On further review of this case, I believe it unwise to label it as an example of "congenital idiopathic hypertrophy" of the heart. There certainly was hypertrophy, with a history of congestive failure and a cardiac death, but there was no evidence of any heart trouble until the severe illness at five. To my mind, that illness in some manner, toxic or otherwise, which we do not understand, quite probably injured the heart. In view of the intestinal symptoms and fever during the severe acute illness at the age of five, when the heart trouble seemed to begin, it has been suggested that an acute trichinous invasion of the body may have set off the heart trouble.

Dr. Mallory: I think in that case we should have found encysted trichinae, since once encysted they never disappear.

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"THE AMERICAN WAY OF LIFE"

PHRASES play an important part in shaping the decisions of our national life, as they flash through the minds of men and women and strike fire from their imaginations. They help to constitute the emotional propaganda by which we form our judgments and see more clearly, or so we hope, the differences between right and wrong.

In 1917, American youth took the eastward passage to "make the world safe for democracy," and if there is too much uncertainty concerning the idealism of the movement, let the doubter see the panels painted by John Singer Sargent in the Widener Library at Cambridge. In 1942, we find ourselves having again accepted the challenge, that in this world of chaos we may preserve "the American way of life."

What is this way of life, of which we are so proud when we find it threatened? Let us call it the opportunity that we, as a people, have seized and preserved of determining our own national destiny and of maintaining certain constitutional rights of the individual. We may not have planned our destiny well or pursued it with vigor, and we may have saddled ourselves with a government by bureaus; but we have done so of our own free will, through our elected representatives, and we still have the power to change it all, should we decide that there is a need.

In the last decade, our freedom has been jeopardized by forces that have required extraordinary opposing measures. Our economic structure has failed us to the point where we have been willing to accept an unbelievable amount of restriction in our daily activities, and in the midst of this depression, in 1931, a wave of aggression was started by totalitarian nations that we, and their other democratic neighbors, lacked the courage to stop.

When military aggression finally assumed epidemic proportions, we became aware of our own danger and began to make the sacrifices that might preserve this American way of life. Voluntarily, we have accepted military conscription; voluntarily, we are giving up our business, our professional lives and our family ties to further the great business of national defense; voluntarily, we have assumed increased taxation, burdensome debts and obedience to dictatorial powers, that, in the end, we might return to our free way of living.

We have, however, no guarantee that our liberties will be returned to us unless we ourselves retain a spirit of freedom that will require their return. Planned economies are still in the making, and it is said that federal socialization of medicine has by no means been permanently abandoned.

It will be well for us all to remember the words of Benjamin Franklin: "You have a Republic, if you can keep it."

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SANATORIUM CARE FOR RHEUMATIC CHILDREN

LAST May, the Sharon Sanatorium closed its doors to patients with tuberculosis. In the fall of 1938, a small group of children with rheumatic fever were placed in an isolated pavilion; this step was owing to the fact, now increasingly apparent, that the ultimate prognosis in this disease is determined not so much by the onset as by the insult of repeated attacks, which are so often precipitated by infections of the upper respiratory tract. The children were protected, so far as possible, from exposure to infections of the upper respiratory tract, lived on open porches, even during the winter, and were kept on more or less of a tuberculosis regime. The preliminary results of this study,* undertaken jointly by the sanatorium and the Children's Mission to Children, were so satisfactory that the directors of the sanatorium became convinced that all the energies of the institution should be devoted to the care of rheumatic children.

In line with this change of policy, the sanatorium has been extensively rebuilt, and equipment for intensive clinical and laboratory studies has been installed. With a bed capacity of forty, divided equally between two buildings, the feature of small units has been preserved.

To provide for the sanatorium care and study of children with rheumatic infection is a new undertaking that should be followed with extreme interest by all members of the medical profession. If the results are confirmatory of those obtained with the preliminary group, it seems likely that a means will be provided for reducing not only the mortality but also the cardiac crippling that is the usual accompaniment of the disease.

*Hubbard, J. P., and Griffin, W. A. Open-air sanatorium care for patients with rheumatic fever and rheumatic heart disease. *New Eng. J. Med.* 223:968-972, 1940.

OBITUARY

HENRY AUSTIN WOOD

1855 - 1942

On February 21, 1942, the long life of Henry Austin Wood, of Waltham, was closed at the age of eighty-six. Born in Upton, Massachusetts, he attended Exeter and Harvard, receiving his medical degree in 1883 and settling in Waltham in 1888. Here he practiced medicine and surgery through the professionally eventful years that followed, becoming one of those remarkable doctors whose friends are legion and whose accomplishments are almost legendary in their native heaths. Of the more than 4000 babies he delivered, probably over half were born in their homes. How many major abdominal operations he performed can hardly be estimated, but many recall protracted periods when they averaged one a day; if a day passed without one major operation, there were usually two or three of them on the day following. This volume of work may be easily surpassed in the activities of many specialists, but it must be recalled that the doctor personally attended to all the practice that generated these deliveries and operations, if one is to appreciate the immensity of Dr. Wood's daily, monthly and yearly rounds. For a quarter of a century, he served on the School Committee; for half a century on the staff of the Waltham Hospital; for many years on many local boards and committees of the city in which he worked and lived and of which he was a part. The remarkable thing, and in his later years the notorious thing, about Henry Wood was his capacity to endure this continual pressure. Younger doctors have frequently embarked on such a life, only to find themselves burned out at the end of a decade. Dr. Wood wore out scores of horses, and later when the Fords came, he wore out the Fords.

The qualities that permit a man to accomplish this are of interest. There must first be mental and physical vigor, but that is not all. Many a vigorous character has made a fair bid for similar accomplishment only to learn what every woman knows: that his wife must contribute quite as much to such a life as he himself. And other men are tripped in their headlong course by their children, or by their inability to secure the faithful ancillary services needed to keep such a practice rolling. Henry Wood was fortunate in all these ways; he attracted faithful servants, like Mary Manning and Frank Christmas; his children never tripped him up; but above all he married a discerning and unselfish person who over the years became equally well known and beloved in her adopted city — Anna Wharton Smith. She, two daughters and two sons survive him. Although it was not the sort of thing he talked about, in his declining years it was a great comfort that the younger son was carrying on his practice in the modern way.

D. O'H.

MEDICAL EPONYM

Murphy Maneuver

On December 18, 1902, Dr. John Benjamin Murphy (1857-1916) described the following diagnostic maneuver, to which his name is frequently given, before the New York Academy of Medicine. His paper appeared in *Medical News* (**82:** 825-833, 1903), under the title, "The Diagnosis of Gall-Stones."

> . . . . The most characteristic and constant sign of gall-bladder hypersensitiveness is the inability of the patient to take a full, deep inspiration, when the physician's fingers are hooked up deep beneath the right costal arch below the hepatic margin. The diaphragm forces the liver down until the sensitive gall-bladder reaches the examining fingers, when the inspiration suddenly ceases as though it had been shut off. I have never found this sign absent in a calculous or infectious case of gall-bladder, or duct disease.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEE ON MATERNAL WELFARE*

Case History: Post-partum hemorrhage followed by sepsis and death

A thirty-nine-year-old primipara was first seen when approximately two months pregnant and was followed for approximately every three weeks until the onset of labor. The prenatal care was excellent. The past history was not remarkable. The heart and lungs were normal, as was the blood pressure. The blood Wassermann reaction was negative. Measurements of the pelvis were said to be normal. Pregnancy was uncomplicated except for grippe at about seven months.

Labor was normal and lasted only six hours, being terminated by a simple forceps delivery. Previous to delivery, the patient had been given two injections of pituitary extract, each consisting of 5 minims. She sustained a first-degree tear of the perineum. There was difficulty in the delivery of the placenta, which was expelled only after the greatest effort. The central part of the placenta was described as pale and had apparently been firmly attached to the uterus. There was no mention of unusual bleeding until two hours after the placenta had been delivered. At this time, the patient was said to have been flowing very freely. Ice, ergot, pituitary extract and morphine were administered. The patient suffered another severe hemorrhage the following morning, and she was given 500 cc. of citrated blood. On the fourth day, a septic temperature developed. Two more transfusions were administered. Death from sepsis resulted on the seventh day. No autopsy was performed.

Comment. The use of pituitary extract in such large doses cannot be condoned. Presumably, although the record does not state this, the administration was delayed until full dilatation. The intelligent use of pituitary extract in minim doses after full dilatation when there is no disproportion is good obstetrics and frequently results in normal delivery. The record makes no note of any difficulty in the delivery, nor did the patient suffer any severe lacerations. Episiotomy was not performed. Most obstetricians prefer episiotomy, with its resultant clean wound, to the ragged laceration that the perineum so often sustains. The description of the placenta leads one to think that some placental tissue may have remained because of a minor degree of adherent placenta, or possibly that a true area of placenta accreta existed. The hemorrhage following so long after delivery, with the description of the placenta, confirms this supposition, and the fact that no subsequent hemorrhage is recorded in no way contradicts it. There is no mention in the record that the uterus was invaded after delivery. A hemorrhage such as that described usually stimulates intrauterine investigation. If the uterus had been invaded and an accreta diagnosed, hysterectomy after transfusion would have been the logical course. If a piece of adherent placenta had been found as the cause of this post-partum hemorrhage, it might have been removed manually. At any rate, in the face of such hemorrhage, it would have been wise to have packed the uterus and, if hemorrhage continued, to consider the advisability of hysterectomy. According to the record, however, no subsequent hemorrhage developed, but sepsis did follow. It is quite possible, in spite of the lack of positive evidence, that the uterus in some way was invaded at the time of the severe hemorrhage. If this was so, the following sepsis may have had its beginning in this manipulation. This death occurred before the era of chemotherapy.

*A series of selected case histories will be published weekly. Comments and questions by subscribers are solicited and will be answered by members of the committee. Letters should be addressed to the secretary, Dr. Raymond S. Titus, 330 Dartmouth Street, Boston.

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TRACEY, MARTIN L., 63 Longwood Avenue, Brookline.
Jefferson Medical College of Philadelphia, 1936.

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NORFOLK SOUTH DISTRICT

DAVIS, ALBERT, 191 Sea Street, Quincy.
Middlesex University School of Medicine, 1936. Sponsor: Benjamin F. Bornstein, 370 Commonwealth Avenue, Boston.

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University of Cincinnati College of Medicine, 1937.

JETTER, WALTER W., 6 Bay View Avenue, Hingham.
University of Buffalo School of Medicine, 1931.

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PLYMOUTH DISTRICT

CHASE, JOHN S., 1004 North Main Street, Brockton.
Harvard Medical School, 1937.

DOUGLASS, HECTOR B., 30 Bedford Street, Bridgewater.
Middlesex University School of Medicine, 1936. Sponsor: Ralph C. McLeod, Goddard Hospital, Brockton.

FITZGERALD, EDWARD F., 430 Washington Street, Whitman.
Georgetown University School of Medicine, 1939.

GOLDFARB, SAMUEL, 19 West Central Avenue, Onset.
College of Physicians and Surgeons, Boston, 1936. Sponsor: Raymond H. Baxter, 6 South Street, Marion.

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Boston University School of Medicine, 1937.

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Middlesex University School of Medicine, 1937. Sponsor: Peirce H. Leavitt, Goddard Hospital, Brockton.

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BLANCHARD, STANLEY W., 411 Marlboro Street, Boston.
Tufts College Medical School, 1905.

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Boston University School of Medicine, 1937.

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Tufts College Medical School, 1939.

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College of Physicians and Surgeons, Boston, 1930. Sponsor: John J. Federkiewicz, 155 Savin Hill Avenue, Dorchester.

DEXTER, LEWIS, 514 Park Drive, Boston.
Harvard Medical School, 1936.

FLYNN, SIMON A., Boston City Hospital, Boston.
McGill University Faculty of Medicine, 1938.

GATEMAN, MURRAY, 505 Massachusetts Avenue, Boston.
Middlesex University School of Medicine, 1934. Sponsor: Max Ritvo, 485 Commonwealth Avenue, Boston.

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Tufts College Medical School, 1939.

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Middlesex University School of Medicine, 1936. Sponsor: William H. Blanchard, Soldiers' Home Hospital, Chelsea.

KARP, ISADORE A., 153 Shurtleff Street, Chelsea.
Middlesex University School of Medicine, 1936. Sponsor: Arthur Berk, 270 Commonwealth Avenue, Boston.

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University of Budapest Medical School, 1913. Sponsor Philip E. Meltzer, 20 Charlesgate West, Boston.

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Duke University School of Medicine, 1936.

PALMIERI, SALVATORE P., 20 Davis Street, Boston.
Tufts College Medical School, 1941.

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University of Vienna, 1906. Sponsor: Joseph Goodman, 375 Commonwealth Avenue, Boston.

SOUTTER, LAMAR, 13 West Cedar Street, Boston.
Harvard Medical School, 1935.

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Tufts College Medical School, 1931.

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WORCESTER DISTRICT

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University of Turino, 1932. Sponsor: Bardwell H. Flower, Worcester State Hospital, Worcester.

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Boston University School of Medicine, 1940.

BUONO, CHARLES L., 98 Beverly Road, Worcester.
Middlesex University School of Medicine, 1934. Sponsor: Joseph P. Mulhern, 390 Main Street, Worcester.

CARTIER, ROLAND R., Rutland State Sanatorium, Rutland.
University of Montreal Faculty of Medicine, 1933.

DURFEE, MARION B., Worcester State Hospital, Worcester.
University of Colorado School of Medicine, 1934.

FAIRBANKS, EDWARD J., 22 Nelson Street, Webster.
Harvard Medical School, 1937.

GALUSZKA, BRONISLAUS A., South Street, Barre.
Tufts College Medical School, 1939.

JACOB, LOUISE H., State Hospital, Westboro.
University of Colorado School of Medicine, 1938.

KELLY, FRANCIS J., Hahnemann Hospital, Worcester.
Hahnemann Medical College and Hospital of Philadelphia, 1941.

KRETZMER, EUGENE, 29 Maplewood Road, Worcester.
University of Munich, 1908. Sponsor: George D. Kaneb, 28 Pleasant Street, Worcester.

LANKENNER, PETER A., 522 Grafton Street, Worcester.
Middlesex University School of Medicine, 1932. Sponsor: Joseph P. Mulhern, 390 Main Street, Worcester.

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Mid-West Medical College, 1934. Sponsor: Paul P. Montag, 200 Highland Street, Worcester.

MULLOWNEY, JAMES P., 71 Jaques Avenue, Worcester.
Loyola University School of Medicine, 1938.

SACHS, BARNEY E., 989 Main Street, Leicester.
College of Physicians and Surgeons, Boston, 1934. Sponsor: Bennet I. Fielding, 60 Franklin Street, Worcester.

SEIDENBERG, DANIEL, 12 Pleasant Street, Spencer.
Kansas City University of Physicians and Surgeons, 1929. Sponsor: James C. Austin, 176 Main Street, Spencer.

TEED, ROY W., 24 Congress Street, Milford.
Middlesex University School of Medicine, 1935. Sponsor: Joseph Ashkins, 36 Pine Street, Milford.

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WORCESTER NORTH

MARNANE, JOSEPH P., 31 Nutting Street, Gardner.
Tufts College Medical School, 1938.

SHAPIRO, MORRIS W., Gardner State Hospital, East Gardner.
Tufts College Medical School, 1939.

WYANT, JOHN A., 101 Prichard Street, Fitchburg.
College of Physicians and Surgeons, Boston, 1936. Sponsor: Alverne P. Lowell, 52 Hartwell Street, Fitchburg.

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CONGRESS ON MEDICAL EDUCATION AND LICENSURE

The thirty-eighth Annual Congress on Medical Education and Licensure held recently in Chicago proved so interesting that, as delegate from the Massachusetts Medical Society, I am presenting certain impressions of the meeting while the various happenings are still fresh in my mind.

On Saturday afternoon, February 14, I attended a long meeting of the Association of American Medical Colleges. Here, for more than five hours, the matter of the accelerated program of medical education was discussed. The upshot of the discussion led to several resolutions, which were passed by that body. Among the most significant were the following:

Approval by the association of the accelerated program as an emergency measure.

Minimum standards for an approved course to include four terms of not less than thirty-two weeks each, and no student to graduate sooner than thirty-six months after his registration as a first-year medical student.

Standards of eligibility to an approved school not to be lowered.

The council of the association was ordered to negotiate with the proper governmental agency for the establishment of a student-loan or scholarship fund, not to exceed $3,500,000 a year, loans or scholarships from such a fund to be awarded on the recommendation of their deans to students needing financial assistance to carry out the accelerated program.

The association did not approve at the present time of asking federal grants to help in the financing of medical education; such aid as was to be sought was clearly in order to help needy students rather than the financial state of any medical school.

Internships were not to be regarded as acceptable when they were of less than twelve months' duration.

No school was to feel any sense of compulsion to attempt an accelerated program. Unless such a program could be carried out effectively by any school without lowering the quality of the medical education offered, it should not be initiated.

Before these resolutions were passed, Dr. Walter Bierring, of Iowa, reported that sixty-three schools had already made plans to proceed on the accelerated program. He gave a careful analysis of individual state-board rulings, which showed the difficulties that certain states will have in modifying their medical registration laws to be compatible with an accelerated program of medical education.

Major Sam F. Seeley, of Washington, D. C., outlined the plans of the Procurement and Assignment Service. A complete account of this has been printed in the February 21 issue of the *Journal of the American Medical Association*, so that I shall not attempt to describe it here. Major Seeley, however, emphasized that the Procurement and Assignment Service will do all in its power to protect hospitals, medical schools and communities from medical deprivation. It is hoped that enough volunteers who are not "essential" doctors will be found to yield an adequate supply of medical officers for the needs of the armed forces. He described the methods to be pursued for grading "essential" doctors and for the deferment from active service of commissioned officers who at present are doing work essential to civilian needs. The success of the plan will hinge on the readiness of all physicians to supply data by filling out in detail the form that will soon be sent them, and on their unselfishness in being ready to undertake any type of work assigned them.

Dr. Willard C. Rappleye, of New York City, added significantly to the afternoon's discussion. He traced the development of the "accelerated plan" and pointed out that it had come into being largely because the Selective Service System realized the urgent need of physicians for military and civilian use and failed to see the necessity in times like these of the long vacation for medical students hitherto in vogue. On the other hand, he reiterated that although the emergency must be met, standards of medical education must not be lowered.

As a concluding item on the program, Dr. Fred Zappfe, of Chicago, called attention to the present shortage of microscopes for the use of medical students. He suggested that a survey of the supply of microscopes be made by each school and that "microscope-loan pools" be established in different schools through their alumni associations. Alumni owning microscopes and not using them regularly could do a great service in lending their instruments to students, because new ones are almost unattainable. By such means, an adequate number of microscopes, properly conditioned, could be made available to students at relatively little expense. He also pointed out that a shortage of blood-counting equipment and blood-pressure machines may develop.

On Sunday, February 15, I attended the meeting of the Council on Medical Education and Hospitals of the American Medical Association. The council supported the attitude of the Association of Medical Colleges toward the accelerated program of medical education, emphasizing, too, that the decision of a medical school to initiate such a program was not compulsory and should be reached only after a comprehensive survey of its own ability to speed up its program of education without deterioration of the quality of its medical instruction.

On Sunday evening, I attended a joint meeting of the Council on Medical Education and Hospitals and the Advisory Board of Medical Specialties. This provoked interesting points of view. It seemed fair to presume that specialists would be of use to the armed forces and would continue to be of use to the civilian population. Therefore, new specialists should be developed to meet these needs, and the idea of continuing training for properly equipped young physicians should not be abandoned. Various suggestions concerning how this might be best accomplished were debated.

The Annual Congress on Medical Education and Licensure was held during the following Monday and Tuesday, February 16 and 17. All the papers that I heard were interesting, although I was especially impressed with the one on Monday morning by Professor William B. Munro, of Pasadena, California. His title was, "The War, the Colleges and Federal Aid." His viewpoint seemed to be that we had already developed in the United States, through our plan of wide collegiate education, a good many college graduates who by no means were well educated. If governmental funds were to be made readily available to colleges, he feared that emphasis thereby might be laid on the quantity of students accepted rather

than on the quality of the education that they received, and this to him seemed unwise. He stated that scholarships with which to assist needy students, should federal aid become available for this purpose, were better than loan funds because they could more easily be awarded on the basis of scholastic aptitude and need rather than on need alone. He concluded by saying that, in his opinion, endowed universities should do all in their power to maintain their independence. Evidently, he feared that too much in the way of governmental subsidy of education might lead to restriction rather than to liberality in thought and study.

Monday afternoon was devoted to a discussion of war problems. Colonel George F. Lull spoke for the Army, Surgeon General Ross T. McIntire for the Navy, Surgeon General Parran for the Public Health Service, Colonel Leonard G. Rowntree for the Selective Service System, and Dr. Irvin Abell for the Committee on Medical Preparedness of the American Medical Association.

The need for doctors was emphasized again. The type of training that would develop most desirable officers for the Army and Navy was described. What the Selective Service System has already accomplished was brought out very graphically. And, finally, that all doctors will be useful to the country in some manner was made very plain.

Dr. Harold S. Diehl, of Minneapolis, concluded the session with an interesting paper on "The Role of Medical Schools in the War." It seemed clear to him that, of necessity, medical students must play an increasingly significant part in the country's history. Already certain features of military medicine have become of great importance to the country's needs, and he suggested that medical educators might well begin to point their teaching in the direction of giving medical students a better knowledge than heretofore of these new matters. He suggested, for example, that "first aid" should be taught to first-year medical students. The emergency care of wounds and minor sepsis deserves more systematic attention than this topic often receives in the normal peacetime curriculum. Psychiatry is rapidly becoming of noteworthy significance. Public health, tropical medicine, dermatology and venereal prophylaxis are general subjects that Army officers report as being inadequately covered in the undergraduate courses, as evidenced by lack of knowledge in these matters on the part of newly inducted officers. Finally, aviation medicine should be taught more emphatically, especially from the viewpoint of its physiologic aspects. Aviation medicine offers an important field of endeavor to competent young men with sufficient basic knowledge and training to enter it successfully.

On Monday evening, I attended the dinner of the Federation of State Medical Boards. This ended in a prolonged discussion of the accelerated program, entered into by state health officers from all over the country. The conflict of certain medical registration acts toward such a plan was again described. Finally, the federation expressed itself as being in sympathy with the move toward an accelerated program of medical education, regarding this, however, as a war emergency measure rather than as a permanent change in policy. It was stated that, when necessary, state laws on medical registration should be so modified as to make acceleration permissible during the period of emergency.

The program on Tuesday dealt less with matters pertaining to war medicine.

The Congress on the whole was the most significant one that I have attended. I came home feeling that the medical profession is playing a major part in national defense, and is solving the many problems with which the country is now faced in a manner both creditable and intelligent.

REGINALD FITZ

"THE INNER WORLD OF THE CHILD"*

The child's inner world is full of color and feeling. It is simpler than the world we know and sometimes happier. The child has little information, but a great deal of curiosity. This curiosity is usually an expression of his need for learning. Sometimes, it can be an expression of the child's anxiety or his fears.

The child imitates and acquires. He learns by doing and imagining. He wants to love and be loved. He is only vaguely aware of what adults see in the world around them. He knows the difference between pain and pleasure, having and not having, coming and going away; death, to him, means little more than going away.

The child has many needs of which he may not be aware, and often he finds it hard to make his wishes clear. Thus, he must depend on the adults around him to understand and take care of his needs in a healthy way.

Next to himself, in the inner world of the child, the mother and father — or their substitutes — are most important. They are king and queen of the child's court, the mountains on both sides of his valley. In the simplicity of his inner world, the parents loom like giants or heroes toward whom he has affectionate and anxious feelings. Parents often forget this; some never know it.

Between the external situation of his life and his internal emotions, the child has to strike some sort of balance or make some sort of adjustment. But there is always a gap between what he wishes and what he gets from reality, and that gap, or deficit, is what fantasy fulfills. Whether it is made up in play, by story-telling, in little private dramas or in daydreaming, his fantasy life is always significant. It shows the direction in which the wishes go, as well as the failure of environment to provide what the child wants. The fantasies of a child are naturally fantastic. When they become real, they may be the accomplishments of the world. Fantasies and only fantasies explain the pyramids of Egypt, or an empire. Sometimes, the child's dream is more important to him than reality, or anything else in the world, and far more beautiful. It is natural for him to have daydreams. Don't laugh at him, or you may kill the goose that lays the golden egg. At some time or other in childhood, every girl is Cinderella or Snow White, and every boy is George Washington or the Dutch lad who held his finger in the hole in the dike.

The child does not understand the outer world as we understand it, but he understands it by what is called *intuition*, especially how it feels toward him; for example, it is said that children and dogs sense instantly how strangers feel toward them. Usually, children know without being told how those around them feel. Children are not so unaware of things as they may appear to be; as Wordsworth said, they are "not in entire forgetfulness, and not in utter nakedness."

If there is hostility or financial tension in a home, children sooner or later are aware of it. If there is pain or suffering or insecurity around them, they know about it. If there is conflict between parents, children sense it; they do not need to be told. They soon learn to adjust them-

*A "Green Lights to Health" broadcast given through Station WAAB by Dr. Merrill Moore on Saturday, December 20, and sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

selves to it or take advantage of it. On this account and for the good of the child, if for no other reason, parents should present a united front to their children, and, whenever possible, should keep in fair agreement. Otherwise, the child quickly learns to play one off against the other, and from the point of view of discipline such a child "sits down between two chairs." That is how he learns to feel conflict in himself, and how to be undisciplined and disorganized, for a child takes on a part of the personality of each parent.

If a parent is disturbed or hurt, this can be reflected in the child's conduct, play or schoolwork. One teacher who has what I call "emotional intelligence" said to me recently, "When a child we know to be bright begins to do poor work, we always look to see what is wrong at home." This point has been emphasized by Dr. Douglas A. Thom, director of the Habit Clinic for Child Guidance in Boston. Dr. Arlie V. Bock, who has made similar observations in his study of normal college students at Harvard, believes that when students who have good minds fail to do good work, it is usually the reflection of some inner emotional problem or a disturbance at home.

The child is aware of his own body, which is very important to him. He begins early to understand what he is told about it. He remembers part of what he is told, and sometimes he can use that information with a kind of inborn intelligence.

Children recognize and appreciate the truth. They may ask either direct or indirect questions. It is essential for the parent to be aware of the possibilities of indirect questions, and to try to satisfy the child's curiosity, but the parent should not be overconscientious and answer questions that have not yet arisen in the child's mind. This confuses and overstimulates the child. A single question may need to be answered many times, since the child's growing understanding gradually takes in different phases of the answer.

Very close to the heart of a child is his awareness of his own sex. This should be treated with conscious respect. Adults can do a great deal to disturb the emotional development of some children, through misinformation or teasing. I know one person whose usual approach to a child (if it is a boy) is to say, "Oh! what a pretty little girl!" This always gets a rise out of the child, which the adult enjoys. But sometimes the child is mortified or enraged, and this may be only fuel to the flames of vanity in a person who likes to show off or be cruel.

It is normal for children to exhibit and, in childish ways, to be unkind. But as they grow up and learn other pleasures, normal people tend to give up childish behavior. Small boys are naturally rough and aggressive, but modern nursery-schools are doing a great deal to show parents how to deal with these impulses in a constructive way. Well-run nursery schools have much to offer parents as well as children, and a tactful, intelligent kindergarten teacher can do much to help the child bridge the gap between home and primary school, and thus make happy adjustments to the demands of a group of children of his own size and age and general capacity.

Next to kindness, the child's greatest need is to be told the truth — to be given information he can use — again and again. This is how the child gains intellectual security. After the truth, the child needs fairness in all dealings with him. Fair treatment means more to the child than many people realize. Some of the most violent deeds in the world have been done as the result of a hurt or defeated sense of fair play. Children understand that. A woman told me once that Lincoln's Gettysburg address always brought tears to her eyes when she heard it. I asked her why. She said, "Because it makes me feel he was so kind, and so *fair*!"

To boil it down to a few words, then, What does a child need to make his inner world well organized, his efforts well directed, his expenditure of energy balanced and his whole personality free from conflict and unnecessary suffering? What can he acquire from the world around him, including his parents? Ideally, a good constitution and healthful living conditions, an opportunity to play and to learn to work. Practically, the child needs what the adult needs, only more of it. There is no limit to the amount of affection a child can take, short of spoiling him. He needs to be praised, guided and reassured. Parents should try to instill self-reliance and not teach children to fear, as some silly people do. A child needs science, in its elements, and art. Show a child a mineral, a sea shell, a fossil, an insect or an animal. Notice the pleasure it gives him. Or show him a picture, a statue, a poem or a place, and tell him the story of it. He will enjoy that and learn from it. A child's attention is easy to gain but not so easy to hold, for his span of attention is short. However, all the time you are speaking to a child his brain is recording what you say, and his spirit is being fed by it.

All the time you are with a child you are giving him facts, experiences, good or bad suggestion, and beautiful or ugly images. It is out of this material that he builds the inner world of his mind. In this inner world, his self (or ego) lives. It is a self that the world may later come to know happily or unhappily. It is this self that has to deal with his emotions and his conscience and with the outside world. For a child to grow and function best and cope with what he has to meet to grow up and go through life, he needs health, protection, security, affection, guidance and discipline, a chance to develop independence and real opportunities to be happy and useful.

DEATH

GOODWIN — HAROLD C. GOODWIN, M.D., of Springfield, died March 1. He was in his sixty-fourth year.

Born in Peabody, Dr. Goodwin received his degree from Dartmouth Medical School in 1900. He was division examiner of prisoners for the Commonwealth of Massachusetts, and was a member of the staffs of the Mercy and the Wesson Memorial hospitals and of the Mary Lane Hospital in Ware. He was a fellow of the Massachusetts Medical Society and the American Medical Association.

He is survived by his widow and two sons.

MISCELLANY

RESULTS OF THORACOPLASTY

The number of persons whose pulmonary tuberculosis has been arrested through the aid of thoracoplasty is steadily increasing. They will require medical surveillance for the rest of their lives. They are not immune to other diseases. Since any doctor may be called on for advice, it is desirable that all members of the profession be familiar with the changes in the thorax that are brought about by thoracoplasty. An abstract of a recent discussion (Overholt, R. N. Permanent collapse therapy in pulmonary tuberculosis. *J. A. M. A.* **117**:1681–1687, 1941) follows:

In advanced tuberculous disease, tissue destruction and cavity formation have taken place, the elasticity of adja-

cent segments of the lungs is frequently decreased, and the volume of the healthy lung is so reduced that it is incapable of filling the unyielding thoracic space. How can healing take place under such conditions?

Pulmonary cavities must be closed and must remain closed. Fibrous tissue must not tear under the strain of ordinary thoracic movement, to ensure against reactivation or hemorrhage. The scar must be solid, but at the same time its contraction should not pull thoracic viscera out of position to the extent that cardiorespiratory function is impaired. Certain patients are fortunate enough to make these adjustments spontaneously, arrest the process, and enjoy moderate activity without reactivation. For a second group, the problem has been solved by an adequate pneumothorax (or other temporary measure), which is maintained indefinitely with safety. There is a third group to which thoracoplasty is not applicable; patients who have disease so extensive and so distributed as to make it technically impossible to bring it wholly under control by any single method or combination of methods must be excluded from this discussion.

A significant proportion of tuberculous patients do not fall into the foregoing categories. The health of patients in this fourth group can be restored with surgical help. They are those patients who suffer from advanced disease with irreparable pulmonary damage.

A discrepancy exists between the volume of healthy lung and the volume of the thorax. Temporary measures have failed or present no reasonable chance of being effective. They have an equivalent of two healthy pulmonary lobes, the two on one side or one on each side. Preferably, the disease is stable. The thoracic cage can be refashioned and the diseased lung released from its anchorages. The permanently altered position of the chest wall provides a permanent collapse.

Modern thoracoplasty accomplishes the following: fibrous tissue is released, permitting cavity closure; pulmonary tissue, which has been partially damaged but not totally destroyed and whose elasticity has been impaired by fibrosis, is relaxed; limitation of motion is imposed on the diseased lung; the collapse of the lung can be made highly selective, with conservation of healthy portions of the lungs; and disturbances due to distortion of the thoracic viscera, such as upward displacement of the lower lobe and lateral displacement of the heart and great vessels, are corrected.

All these readjustments are common accomplishments of a free pleura pneumothorax and thoracoplasty. In addition to these considerations, there are added benefits that are unique for thoracoplasty: thoracoplasty adjusts the thoracic volume so that it comes to equal the volume of the healthy lung — in other words, the functionless portion of the lung is placed under permanent control; the risk of tuberculous or mixed empyema developing in an artificially maintained air space is eliminated; and the risk of spontaneous pneumothorax on the side of treatment is greatly lessened.

The ultimate fate of patients treated by thoracoplasty cannot be determined until more time has elapsed. However, a preliminary study made of patients treated successfully by thoracoplasty and discharged with the consent of their medical advisers is most encouraging. Of 107 patients discharged five or more years, 101 are living, 3 died of tuberculosis and 3 died of other causes. Of 315 patients discharged under five years, 308 are living, 1 died of tuberculosis, and 6 died of other causes.

Although exactly comparable end results are impossible to find, it is fair to assume that the severity and extent of the process from which the groups under discussion suffered were more threatening than those of the average patient undergoing sanatorium treatment. Yet they seem to fare better, for a study of 6906 patients discharged alive from various sanatoriums in this country revealed that only 60 per cent of those discharged with consent were living after a period of five years.

In support of the belief that permanent collapse increases the chances for lasting results, the author quotes Roberts from the *Brompton Hospital Reports* (1936) as follows: "It is shown that the chance of surviving five years in B 3 cases [not defined in the article] treated without collapse was 23.7%; with pneumothorax, 55.3%; and with thoracoplasty, 66.6%. Thus, the expectation of living five years is approximately three times as great in cases submitted to thoracoplasty as in the average B 3 case."

A questionnaire sent to patients treated by thoracoplasty and discharged with consent brought 293 replies. The great majority considered themselves well and were glad they had had a thoracoplasty; 83 per cent were able to work; and 70 per cent declared they had no limitation of arm or shoulder motion. Many letters that accompanied the questionnaire replies stated that the scar and changes in contour of the chest constituted a small price to pay for restoration of health, and many stated that their only regret was that the operation had not been performed sooner.

Several refinements of thoracoplasty have been made since de Cerenville performed the first thoracoplasty in 1885. These include lung palpation at operation, specific mobilization and the liberation of anchoring structures over areas of disease, preservation of periosteal elements and subtotal scapulectomy to minimize deformity in partial thoracoplasty. — Reprinted from *Tuberculosis Abstracts*, February, 1942.

CORRESPONDENCE

STUDY AND TREATMENT OF CONTAMINATED WOUNDS AND BURNS

To the Editor: The Surgical Service of the Massachusetts General Hospital has entered into a contract with the Office of Scientific Research and Development of the United States Government for the study of contaminated accidental wounds and burns and their treatment. The value of the study will be increased if a large number of compound fractures, severe lacerations and extensive burns can be obtained. Toward this end the hospital wishes to notify physicians that facilities will be made available for the transportation and admission of suitable cases on the recommendation of the attendant physician. Ambulance service and admission may be secured through the Admitting Office of the Hospital (CAPitol 4300).

N. W. Faxon, M.D., *Director*

Massachusetts General Hospital
Boston

BOOK REVIEW

A Manual of Bandaging, Splinting and Strapping. By Augustus Thorndike, Jr., M.D. Philadelphia: Lea and Febiger, 1941. 144 pages, with 117 engravings. $1.50.

This is a very handy reference work for those who wish to apply a neat dressing or bandage of any kind. Most of the book is devoted to well-drawn illustrations, which require very little explaining. It should prove popular at this time to the hundreds of people who have suddenly developed an active interest in first aid.

(Notices on page x)